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Chapter 4: Vascular Wounds

Stephanie Woelfel,; Christian Ochoa; Vincent L. Rowe

INTRODUCTION

CHAPTER OBJECTIVES

At the end of this chapter, the learner will be able to:

1. **Relate the pathological changes in the vascular anatomy to the formation of arterial and venous wounds.**
2. **Differentiate between arterial and venous wounds.**
3. **Perform a vascular screening and interpret noninvasive vascular studies for arterial and venous disorders.**
4. **Use the information obtained from vascular studies to develop a plan of care for arterial wounds (before and after surgery) and venous wounds.**
5. **Determine when and if surgical intervention is needed for patients with arterial and venous wounds.**
6. **Select the appropriate compression therapy for patients with lower extremity vascular wounds based upon vascular studies.**

The vascular system is an intricate system of arteries, veins, and lymphatic vessels designed to transport the blood from the heart to the core and peripheral tissue, providing tissue with the oxygen and nutrients necessary to sustain life, and from the same tissue back to the heart and lungs for recirculation (**FIGURE 4-1**). An interruption to blood flow in any one or more of the vessels can cause significant and critical pathologies that result in integumentary changes, wounds, or impaired healing. If the pathology is in the arterial system, the wound is termed *ischemic*; if it is in the venous system, it is termed *venous*. Both types have very defining characteristics and predictable vascular study results that are used to determine the optimal plan of care for the individual patient. This chapter focuses on the pathophysiology, prevention, and treatment of arterial and venous wounds; lymphatic disorders are discussed in Chapter 5, Lymphedema.

FIGURE 4-1

Anatomy of the arterial and venous circulatory systems The circulatory system consists of the cardiac, arterial, venous, and lymphatic systems. The arterial system is further delineated into the macrocirculation (arteries large enough to be named) and microcirculation (capillaries and arterioles too small to be named). The lymphatic system is illustrated in Chapter 5.

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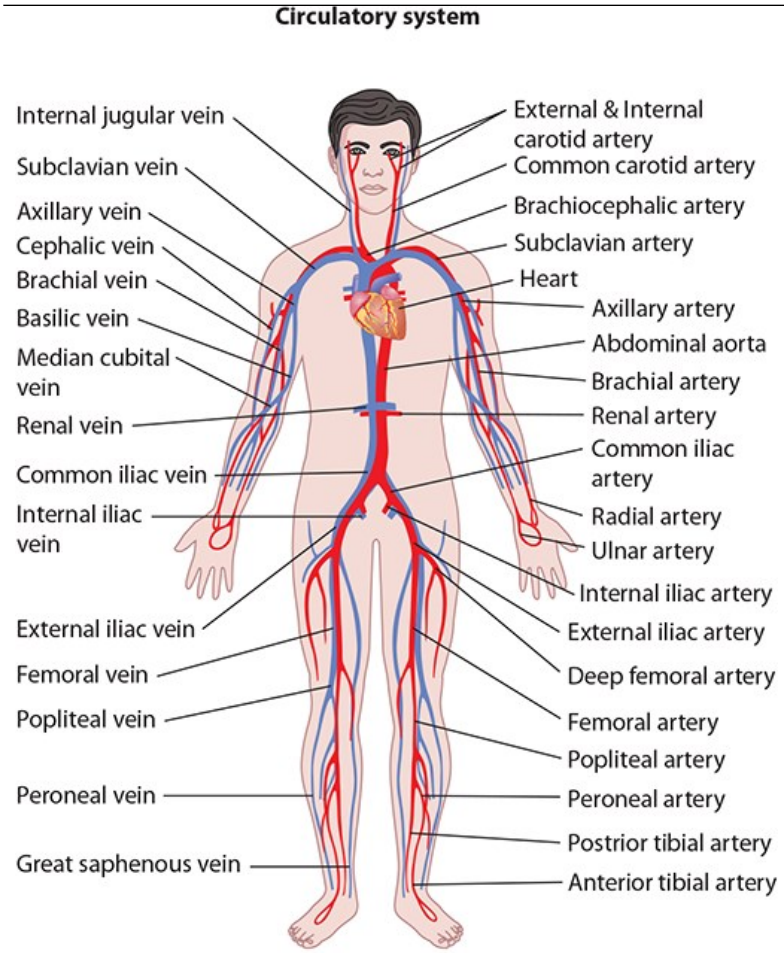

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Vascular diseases such as peripheral artery disease (PAD) and chronic venous insufficiency (CVI) cause the majority of lower extremity wounds; the majority of arterial wounds are caused by PAD. The clinical spectrum of PAD ranges from asymptomatic disease to mild claudication, to tissue loss or gangrene of the foot or lower extremity. When patients with PAD have an ulcer or gangrene of the lower extremity, it is termed *critical limb ischemia* (CLI). The major cause of CLI is a reduction in distal tissue perfusion below the resting metabolic requirements usually associated with atherosclerosis; however, other conditions may cause wounds that appear to be arterial or ischemic (**TABLE 4-1**). Diabetes mellitus (DM) is one of the most serious and prevalent of these disorders. The combination of DM and PAD may lead to foot ulceration or gangrene, which may result in amputation. The overall risk of amputation is 15 times higher for patients with DM than in those without diabetes. The reason for the increased amputation rate is related to the complex pathophysiology of neuropathy and ischemia in the diabetic foot.[1]

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TABLE 4-1

Differential Diagnosis of Lower Extremity Wounds

Inflammatory disorders
- Granuloma annulare
- Necrobiosis lipoidica
- Pyoderma gangrenosum
- Sweet's disease

Malignancy
- Malignant transformation of long-standing ulcer (Marjolin's)
- Metastatic malignancies
- Primary skin malignancies
- Ulcers associated with hematologic or internal malignancies

Pressure ulcers

Infectious disorders
- Bacterial (*Pseudomonas*, staphylococcal scalded skin syndrome, streptococcal necrotizing fasciitis)
- Fungal (blastomycosis, chromomycosis, Madura foot)
- Mycobacterial (*M. leprae, M. tuberculosis, M. ulcerans*)
- Parasitic (Chagas disease, leishmaniasis)
- Viral (herpes simplex, herpes zoster)

Trauma (burns, postsurgical trauma)

Bites (eg, dog, snake, spider)

Medication-related ulcers
- Drug reactions leading to blisters and large-scale wounds (erythema multiforme, Stevens–Johnson syndrome, toxic epidermal necrolysis)

Autoimmune disorders
- Blistering (epidermolysis bullosa, pemphigoid, pemphigus)
- Nonblistering (dermatomyositis, lupus, rheumatoid arthritis, scleroderma)

Atherosclerotic arterial ischemic ulcers

Nonatherosclerotic ischemic ulcers
- Hypertensive ulcers (Martorel's)
- Sickle cell disease
- Thromboangiitis obliterans
- Vasculitis
  - Churg–Strauss syndrome
  - Henoch–Schönlein purpura
  - Leukocytoclastic vasculitis
  - Microscopic polyangiitis
  - Polyarteritis nodosa
  - Urticarial vasculitis
  - Wegener's granulomatosis
  - Embolic (cholesterol emboli, hyperoxaluria)
  - Thrombotic
- Antiphospholipid antibody syndrome
- Disseminated intravascular coagulation, purpura fulminans
- Heparin necrosis
- Homocystinuria
- Livedoid vasculitis
- Polycythemia vera
- Thrombotic thrombocytopenic purpura
- Vasospastic/Raynaud's disease
- Venous ulcers

Neuropathic disorders
- Diabetes
- Leprosy

The pathologies listed in this chart can cause wounds that appear similar to wounds caused by peripheral arterial occlusive disease. Making the correct differential diagnosis is imperative because treatment of the underlying disorder is the first step toward successful wound healing. Some of these disorders are discussed further in Chapter 8.

The presence of any lower extremity wound requires an evaluation of vascular status since it will determine the ability of the wound or surgical incision to heal. Healing of a foot wound may take months despite undergoing revascularization procedures. The presence of CLI in a patient is significant not only for potential limb loss; it is also an independent risk factor for cardiovascular morbidity and mortality. The goal of taking care of these patients is to assure that the maximum amount of arterial perfusion is present to help in the healing of these wounds. There is a high rate of recidivism and subsequent amputation when the treatment of the vascular insufficiency is not performed in a timely manner.

ARTERIAL WOUNDS

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Epidemiology

Between 2000 and 2010, the number of people with PAD has increased by nearly 25%. An estimated 202 million cases of PAD currently exist worldwide. Of these, 20–40 million cases will likely have intermittent claudication and 100 million will have atypical lower extremity symptoms. Forty-five million of the 202 million people with PAD will die from coronary or cerebrovascular disease during a 10-year period.[2]

Overall, the rates for PAD are similar for men and women.[3] The prevalence of PAD rises with age, increasing to over 20% in individuals over 80 years old.[4] Regardless of symptoms, PAD can significantly impact a patient's quality of life, as even patients with asymptomatic PAD demonstrate impaired lower extremity function, greater mobility loss, and quicker functional decline than people without PAD.[2] The true incidence of CLI is unknown; however, some studies have estimated around 500,000 to 1 million new cases occur each year.[5] A similar incidence of 400 per 1 million population per year was found in a national survey of the Vascular Surgical Society of Great Britain and Ireland.[6] The majority of individuals who have CLI with ulceration or gangrene also suffer from DM. It is estimated that up to 70% of all lower extremity amputations are diabetes related and the majority of those patients present with a foot ulcer that develops into an infected limb or gangrene.[7,8] Almost 50% of those patients who undergo amputation for CLI will require amputation of the opposite limb within 5 years.

The economic impact involved in the care of patients with vascular wounds is staggering. The annual cost in treating lower extremity ulcers is around $25 million and the individual cost for treating a foot ulcer can be as high as $28,000.[9] In England, the cost of care for an individual with a leg ulcer was around $130,000 annually.[10] Included in the estimates are physician visits, hospital admissions for wound debridement or surgical revascularization, rehabilitation, and wound care supplies.

The prevalence of PAD *with intermittent claudication* among males and females is between a ratio of 1:1 and 2:1. This ratio increases when an ulcer or gangrene is present to at least 3:1. The prevalence of CLI increases substantially as the population ages.[11]

The natural progression of CLI is limited since published studies include performance of some revascularization procedure. However, there are some patients for whom an operation is prohibitive due to multiple comorbidities or limited life expectancy. A recent study by Marston et al. followed a cohort of patients with CLI who presented with uncomplicated limb ulcers.[12] Revascularization was not performed due to medical comorbidities or anatomic considerations that did not allow surgical intervention. A total of 142 patients were followed for 1 year with the primary endpoint being major limb amputation. During the study, the wounds were treated with a specific protocol that emphasized pressure redistribution, debridement, infection control, and moist wound healing. The limb loss or amputation rate for these patients was 19% at 6 months and 28% at 12 months. Interestingly, complete wound closure was achievable in 25% at 6 months and 52% at 12 months. The only significant factor that affected wound closure was the initial size of the wound.

Arterial insufficiency not only causes wounds, it also portends negatively on the patient lifespan. The most common cause of death in patients with severe PAD or CLI is coronary artery disease. Depending upon the severity of the CLI, patients with resting pain have more than 70% mortality rate at 5 years. Even though this patient population may have a high mortality rate and are often treated in a palliative manner, conservative care can help alleviate pain and/or heal wounds.

Pathophysiology

Risk Factors

The most common causes of PAD are arteriosclerosis and atherosclerosis (**FIGURE 4-2**). Risk factors for the development of these pathologies include age, smoking, diabetes mellitus, hypertension, hypercholesterolemia, dyslipidemia, family history, and obesity. Prevention and treatment of arterial wounds depend upon adequate identification and management of these risk factors.

FIGURE 4-2

Atherosclerosis Atherosclerosis is one type of arteriosclerosis in which fatty plaques composed of low-density fatty acids erode the artery wall with resulting deposition of macrophages and white blood cells. The plaque increases in size over a period of time, causing a gradual decrease in the size of the arterial lumen and reduced blood flow to the distal tissues. The critical level is occlusion of more than 80% of the arterial lumen. In addition,

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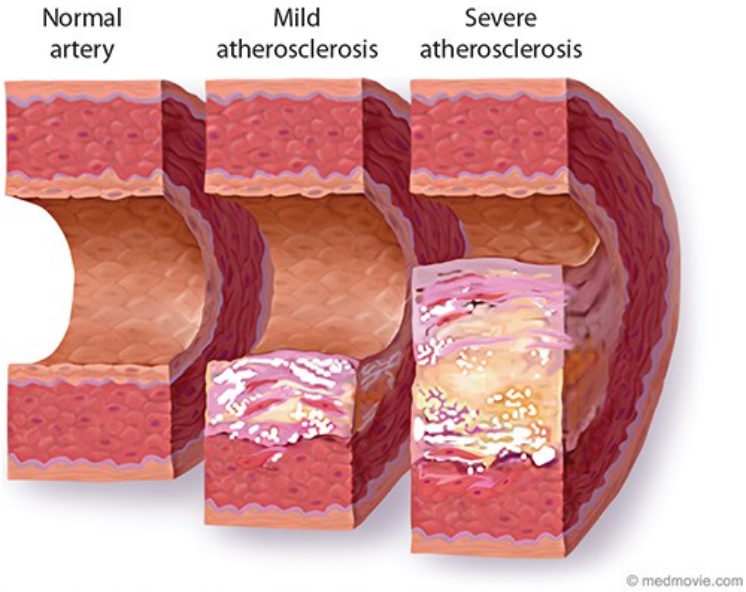
particles of plaque can break away, migrate to smaller arteries or arterioles (termed *thromboemboli*), and also cause tissue ischemia. (Copyright Medmovie.com with all rights reserved.)



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Macro- and Microcirculation

Occlusion of the arterial system can occur in either the *macrocirculation* (defined as those arteries large enough to be named or with a diameter more than 0.5 mm) or the *microcirculation* (defined as the arterioles and capillaries too small to be named or with a diameter less than 0.5 mm). Tissue ischemia that leads to lower extremity wounds tends to occur more in the presence of large vessel or mixed disease. In addition to PAD, the arterial vessels can be occluded by a thrombus or emboli (eg, after surgery or trauma).

Critical Phases of Ischemia

In the early stages of PAD, the circulatory system compensates by establishing collateral circulation around the occlusions in order to maintain peripheral blood flow. The *first critical phase* of the disease occurs when the collateral circulation is insufficient for the metabolic needs of the affected extremity; therefore, the limited blood supply is shunted to the muscle arteries where flow resistance is low instead of to the skin where resistance is high. A wound caused by trauma (eg, a blister from poorly fitting shoes, a skin tear, or a cut from poor foot care) during this period of decreased skin perfusion will heal more slowly than normal, or will be more likely to become infected and fail to heal. The non-healing wound may be the first indication that a patient has PAD.

Because exercise increases the muscle oxygen demand, and thereby its blood volume requirements, a *second critical phase* occurs when activity or exercise causes relative ischemia and pain. Intermittent *claudication*, the term for the symptoms of muscle pain and cramping that occur with exercise, is a second indication that the patient may have PAD.

When the PAD becomes severe, the *third critical phase*, the patient will probably experience resting pain, gangrene, and non-healing wounds in the extremity below the occlusion. During this phase, the patient may exhibit dependent leg syndrome, a position that allows gravity to assist blood flow to the distal extremity and thereby alleviate some of the pain. The patient frequently complains of rest pain during the night, a result of lower blood pressure and subsequent diminished flow to the lower leg and foot.

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CLINICAL CONSIDERATION

Symptoms similar to PAD may be caused by lumbar spinal stenosis. A person with ischemic pain will feel relief with cessation of activity, whereas a person with spinal stenosis claudication will feel relief only with a change of position, for example, standing to sitting or sitting to lying. Determination of whether gluteal/lower extremity pain is due to spinal stenosis or due to ischemia and arterial insufficiency to the painful muscles is important for effective and appropriate medical management.

Etiology

Impairment of blood flow can occur acutely (trauma or thrombosis) or chronically (atherosclerosis or arteriosclerosis). Both acute and chronic arterial insufficiency at any level (arteries, arterioles, capillaries) can lead to the formation of lower or upper extremity wounds. Reduced capillary flow (also called *small vessel disease*) is observed most frequently as a result of diabetes.

Obstruction of arterial flow can also be classified as anatomic or functional. Anatomic causes of obstruction include thromboemboli and vasculitis (**FIGURES 4-3A–D** and **4-4**). Functional impairment occurs in conditions such as Raynaud's phenomenon in which abnormal vasomotor function leads to reversible obstruction (**FIGURE 4-5**). In severe cases this may result in ulceration. Other potential causes of impaired arterial flow include upper extremity arteriovenous fistulas and aneurysms (**FIGURES 4-6A** and **4-6B**).

CLINICAL CONSIDERATION

A wound that has the characteristics of arterial insufficiency on the foot of a young person is a red flag that the etiology is probably not arteriosclerosis. Other atypical disorders (eg, Buerger's disease) need to be considered.

FIGURE 4-3

A, B, C. Arterial wounds due to thromboemboli Arterial wounds that are caused by thromboemboli begin as dusky discoloration (as noted in the third toe of **A** and the foot of **B and C**) and progress to dry or wet gangrene (as noted in the fourth and fifth toes of **A**). If the tissue reperfuses as the medical condition improves, milder cases can resolve and healing can progress almost like a bruise healing. The tissue necrosis may become well-defined by a process termed *demarcation* (as noted in the second toe of **D**). Debridement is usually deferred until demarcation is completed unless there are signs of clinical infection; protective measures are recommended to prevent further tissue loss. Keeping the extremity warm facilitates vasodilation, which can help maximize perfusion. **D. Arterial wound due to trauma** Trauma from multiple causes, including surgery, can result in clotting and thrombi that occlude vessels of any size. Distal tissue will become necrotic unless flow is restored emergently.

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FIGURE 4-4

Ischemic wound due to vasculitis Vasculitis, an inflammation of the vessels that results in edema and occlusion, most often occurs in patients who have autoimmune disorders (eg, systemic lupus erythematosus, rheumatoid arthritis, untreated hepatitis). The wounds are exquisitely painful, do not become necrotic like other arterial wounds, and may occur on any part of the body. (See Chapter 8, Atypical Wounds.)

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FIGURE 4-5

Raynaud's phenomenon Raynaud's syndrome is caused by vasospasm of the small arteries and arterioles resulting in ischemic changes such as skin color, numbness, and cold sensations. The attacks can be triggered by cold exposure or emotional distress. Symptoms are bilateral, begin distal and progress proximally, and in severe chronic cases can result in ulceration or gangrene.

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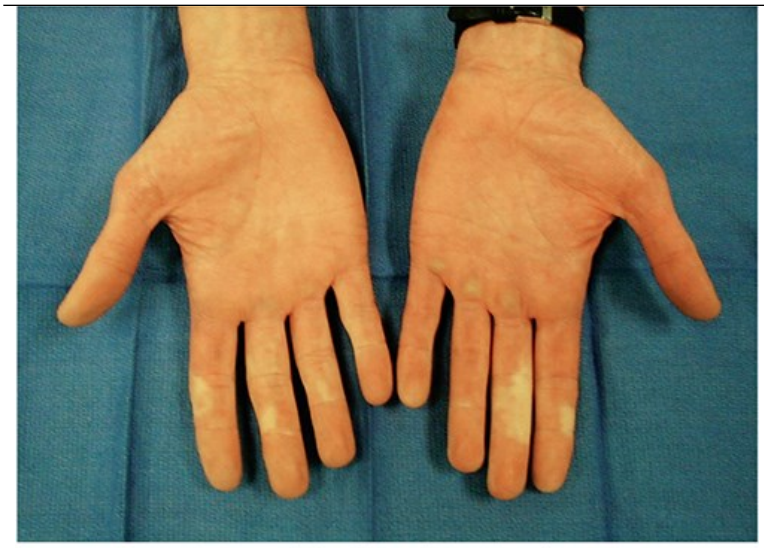

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FIGURE 4-6A

Arterial wound due to AV shunt for hemodialysis Patients who are on long-term hemodialysis and have a history of clotted AV shunts may develop arterial wounds in the fingers as a result of decreased blood flow distal to the shunt.

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FIGURE 4-6B

Arterial wound on the plantar foot due to ruptured aortic aneurysm

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Non-atherosclerotic or vasculitic disorders need to be considered in the patients who present with signs of tissue ischemia in the presence of normal pulses or diminished pulses. For example, thromboangiitis obliterans, also known as Buerger's disease, is a macrovascular disorder. Common in men who are heavy smokers, the pathology is an immune and inflammatory disease of the peripheral arteries accompanied by vasospasm and thrombi in the arterial segments to the feet and/or hands. Occlusion of the arteries causes tissue ischemia with resulting thin, shiny skin and thickened malformed nails. Other symptoms include pain, tenderness, erythema caused by dilated capillaries, thin, shiny skin, and cyanosis caused by deoxygenated blood cells in the interstitium. If the disease exacerbates, the patient is at risk for gangrene or ulceration. Successful treatment must include cessation of smoking (**FIGURES 4-7** and **4-8**).[13] Chapter 8, Atypical Wounds, includes further discussion of ischemic wounds due to unusual pathologies.

FIGURE 4-7

Buerger disease Buerger disease, also known as *thromboangiitis obliterans*, is an immune inflammatory disease of the smaller peripheral arteries. Symptoms can vary from mild pain of the area distal to the inflamed arteries to severe vasospasm with cyanosis or occlusion with gangrene.

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FIGURE 4-8

Radiograph of Buerger disease Arterial radiograph of patient with Buerger disease shows the convoluted, distorted vessels in the distal lower extremity.

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Patient Evaluation

The initial assessment of any wound begins with a thorough history and physical examination, a process necessary to establish a diagnosis of wound etiology. The history includes screening for risk factors of atherosclerosis—especially smoking, diabetes, hyperlipidemia, and hypertension. Patients who have these risk factors need aggressive education and treatment to modify these factors and to encourage lifestyle changes. A thorough vascular medical history of coronary artery disease (angina pectoris or myocardial infarction) and carotid disease (transient ischemic attacks or ischemic stroke) increases the likelihood of PAD because atherosclerosis is a systemic disease and not just localized to a specific part of the body. Assessment of ambulation tolerance (by both patient report and treadmill testing) may uncover mild chronic limb ischemia with intermittent claudication. Other comorbidities, medications, and surgical history are obtained to determine any other contributing factors that may inhibit wound healing.

Clinical Presentation

A thorough wound assessment and information about how the wound began are important in determining the etiology. Refer to Chapter 3, Examination and Evaluation of the Patient with a Wound, for more details on obtaining a subjective history.

The location, size, and depth of the wound are defining characteristics of ischemic wounds, which tend to be small and round with smooth, well-demarcated borders. The wound base is typically pale (lack of arterial inflow), lacks granulation tissue, and may be shallow or deep (**FIGURE 4-9**). Wet or dry necrotic tissue may be present; *wet gangrene* is significant for active infection and must be urgently debrided (**FIGURES 4-10** and **4-11**). Arterial wounds tend to occur at the distal digits, although wounds that occur on other parts of the foot (eg, the malleoli) from pressure may not heal due to arterial insufficiency (**FIGURE 4-12**). Because of the ischemia, arterial wounds are painful and often accompanied by complaints of pain when the feet are elevated, especially at night. Pain that is reduced with leg dependence is indicative of rest pain and is termed dependent leg syndrome.

FIGURE 4-9

Typical arterial wound The typical arterial wound due to PAD is located on the distal digit, has a round punched-out appearance, dry-to-necrotic wound bed, and little or no granulation tissue. The wounds tend to be painful with poor healing potential without restoration of blood supply.

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FIGURE 4-10

Dry gangrene Arterial wounds with dry adhered eschar and no signs of infection are termed dry gangrene.

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FIGURE 4-11

Wet gangrene If the necrotic tissue becomes infected with drainage, detached edges, and wet periwound skin, it is termed wet gangrene.

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FIGURE 4-12

Non-healing pressure ulcer secondary to arterial insufficiency Wounds of other etiologies (eg, trauma, pressure, or venous insufficiency) that fail to heal may be one of the first signs that a person has PAD. If the edges stay adhered with no signs of infection, debridement is deferred or not recommended until perfusion is restored and signs of angiogenesis are visible at the edges. Loose detached edges, as seen in this heel pressure ulcer, may be removed to increase the oxygen perfusion of the periwound skin and decrease the risk of infection.

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Clinical signs of infection such as malodor, exudate, and erythema are noted in order to initiate appropriate antibiotic treatment and proper debridement. The presence of any exudate should also be characterized by the amount and color (refer to Chapter 3). If the wound can be probed to bone, there is a high probability of osteomyelitis (**FIGURE 4-13**).

FIGURE 4-13

Osteomyelitis Osteomyelitis is suspected in any arterial wound that can be probed to bone or that has an edematous "sausage" appearance.

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Other observations indicative of PAD include hair loss, muscle atrophy, atrophy of subcutaneous tissues and skin and appendages, dry, fissured skin, discoloration, and dependent hyperemia (**FIGURE 4-14**).

FIGURE 4-14

Typical appearance of extremity with PAD Extremities that have PAD tend to have thin shiny skin, atrophied subcutaneous tissue and muscles, thick yellow nails, lack of hair, dry fissured skin, discoloration, and dependent hyperemia (termed *rubor of dependency*).

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Wound classification systems exist to stratify the severity of the ulceration and are based on the size, location, amount of tissue loss, presence of infection, and the status of the arterial blood flow. The following three classification systems are specific to diabetic foot wounds: Wagner grading; the University of Texas (UT) classification; and PEDIS or Perfusion, Extent, Depth, Infection, Sensation, which was developed by the International Consensus on the Diabetic Foot (**TABLE 4-2**).[7] A fourth classification system exists for the lower extremity threatened limb and stratifies patients based on **w**ound, **i**schemia, and **f**oot **i**nfection (WIfI) (**FIGURE 4-15**).[14]

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FIGURE 4-15

The WIfI Lower Extremity Threatened Limb Classification The Society for Vascular Surgery developed the WIFI classification system to "permit more meaningful analysis of outcomes for various forms of therapy" in patients who have critical limb ischemia.[14] The system takes into consideration the extent of the wound, the degree of perfusion, and the presence of infection (Compliments of Dr. David G. Armstrong. Used with permission.)

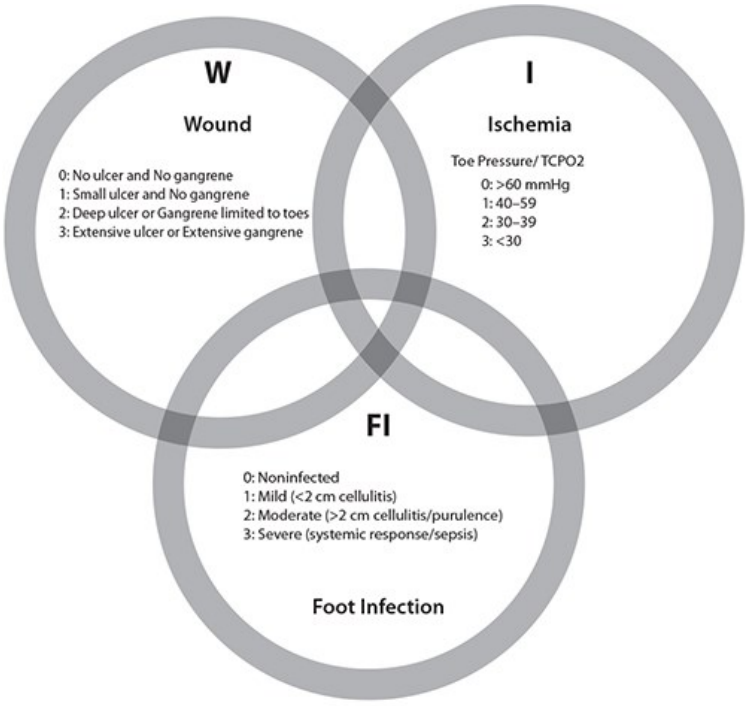

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TABLE 4-2

Classification Systems for Diabetic Foot Wounds

System	Classification	System	Classification
Wagner UT Classification	Grade 0: Impending skin lesion, presence of predisposing bony deformity, or healed ulcer Grade 1: Superficial skin ulcer that does not involve subcutaneous tissue Grade 2: Full-thickness ulcer that exposes bone, tendon, ligaments, or joint capsule Grade 3: Full-	PEDIS Perfusion Extent/size Depth of tissue loss Infection Sensation or neuropathy	1. Wound lacking purulence or inflammation. Uninfected. 2. Presence of more than two manifestations of inflammation (purulence or erythema, pain, tenderness, warmth, or induration), but any cellulitis or erythema extends less than 2 cm around the ulcer, and infection is limited to the skin or superficial or subcutaneous tissues; no other local complications or systemic illness. Mild infection. 3. Infections as grade 2 in a patient who is systemically well and metabolically stable but who has one or more of the following: cellulitis extending >2 cm, lymphangitic streaking spread beneath the superficial fascia, deep-tissue abscess, gangrene, and involvement of muscle, tendon, joint, or bone. Moderate infection. 4. Infection in a patient with systemic toxicity or metabolic instability (eg, fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, severe hyperglycemia, or azotemia).

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thickness ulcer with presence of osteitis, osteomyelitis, or abscess
Grade 4: Gangrenous digit
Grade 5: Gangrene severe enough to necessitate foot amputation
Grade 0: No open lesions; may have deformity
A: Without infection or ischemia
B: With infection
C: With ischemia
D: With infection + ischemia
Grade 1: Superficial wound not involving tendon, capsule, or bone
A: Without infection or ischemia
B: With infection
C: With ischemia
D: With infection + ischemia
Grade 2: Wound penetrating to tendon or capsule
A: Without infection or ischemia
B: With infection
C: With ischemia
D: With

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	infection + ischemia Grade 3: Wound penetrating to tendon or capsule A: Without infection or ischemia B: With infection C: With ischemia D: With infection + ischemia Grade 4: Wound penetrating to bone or joint A: Without infection or ischemia B: With infection C: With ischemia D: With infection + ischemia		

Data from Brodsky JW. Classification of foot lesions in diabetic patients. In: Bowker JH, Pfeifer MA, eds. *Levin and O'Neal's The Diabetic Foot.* 7th ed. Philadelphia, PA: Mosby; 2008:227–239. Armstrong DG, Lipsky B. A guide to new classifications for diabetic foot infections. *Podiatry Today*. Available at: http://www.podiatrytoday.com/article/4838. Accessed October 23, 2013. Bakker K, Apelqvist J, Schaper NC. International Working Group on Diabetic Foot Editorial Board. Practical guidelines on the management and prevention of the diabetic foot 2011. *Diabetes Metab Res Rev*. 2012(suppl 1):225–231.

Non-Invasive Vascular Studies

Pulses

The initial step of any vascular examination is palpation of pulses in the involved extremity that are compared with pulses of the contralateral limb in order to help determine the extent of diminished flow. The palpation of peripheral pulses is affected by the skill of the examiner and the room temperature. Patience is sometimes needed to feel the pedal pulses and the examiner may be misled by “feeling” one's own pulse, especially when using the forefinger which has a strong digital pulse. Strength and interpretation of pulses have two different commonly used grades, thus consistency among team members is needed for accurate communication (**TABLE 4-3**).

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CLINICAL CONSIDERATION

Suggestions for palpating pulses include the following:

Test in a warm room that optimizes arterial dilation, not constriction.

Position the patient in supine with access from both sides of the bed.

Avoid the use of the thumb, which has less discriminating sensation.

Palpate pulses on the contralateral side for an indication of anatomical location.

Encourage the patient to relax so as not to be confused by moving tendons, etc.[15]

TABLE 4-3

Grading Scales for Pulse Examination

1. Scale of 0 to 3+	2. Scale of 0 to 4+
0: Absent pulse 1+: Diminished pulse 2+: Normal pulse 3+: Pathologically prominent pulse, such as the water-hammer pulse of severe aortic insufficiency or if the artery is aneurysmal	0: No palpable pulse 1+: Faint but detectable pulse 2+: Diminished pulse 3+: Normal pulse 4+: Bounding pulse

Data from Hill DR, Smith RB. Examination of the extremities: pulses, bruits, and phlebitis. In: Walker HK, Hall WD, Hurst JW, eds. *Clinical Methods: The History, Physical, and Laboratory Examinations.* 3rd ed. Boston, MA: Butterworths; 1990. Available at: http://www.ncbi.nlm.nih.gov/books/NBK350. Accessed October 20, 2013.

The character of the pulse refers to the upstroke, downstroke, and presence of thrills. In stiffened vessels or in vessels with high outflow resistance, the upstroke or radial expansion of the vessel may be slowed. In the presence of low outflow resistance, such as proximal to a traumatic arteriovenous fistula, downstroke may be significantly reduced. The absence of pulses suggests a proximal critical stenosis or occlusion. After palpation of the pulses, auscultation of the pulses by the examiner permits the detection of bruits, frequently an indicator of an upstream or nearby stenotic lesion. The pulse examination, when correlated with clinical symptoms, helps identify the site and severity of arterial occlusive lesions and indicates when further vascular testing is necessary.

CASE STUDY

Mr. LH is a 74-year-old male admitted to acute care with a non-healing wound on the right lateral heel of more than 6 months duration (**FIGURE 4-16**). Medical history includes type 2 diabetes, end-stage renal disease requiring hemodialysis three times a week, and hypertension. He has been admitted for further vascular studies, pain management, and possible revascularization of the lower extremity. A popliteal artery angioplasty and stent failed to establish blood flow sufficient for healing to occur.

FIGURE 4-16

Case study Right heel pressure ulcer that was first evidence of PAD; size 5.5 cm × 3.3 cm.

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Daily medications include the following:

- Aspirin 81 mg daily
- Clopidogrel (Plavix) 75 mg daily
- Furosemide 40 mg daily
- Isoniazid 300 mg daily
- Sevelamer (Renvela) 800 mg daily
- Sitagliptin (Januvia) 25 mg
- Fish oil
- Vitamin B6

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- Vital signs: normal for age

Laboratory values include the following:

- Hematocrit—31.3%
- Hemoglobin—10.4 g/dL
- Platelets—140,000/mm^3
- Albumin—3.8 g/dL
- HbA1C—8.7
- INR—1
- Prothrombin time (PT)—13.3 seconds
- Partial thromboplastin time (PTT)—30.4
- BUN—50 mg/dL
- Creatinine—8.33 mg/dL

When you enter the room, the patient is sitting in bed with the right leg dangling over the side.

DISCUSSION QUESTIONS

1. Based upon the information in Chapter 3 on evaluation of the patient with a wound, describe the patient's wounds and foot.
2. Classify the patient's wounds using the Wagner, UT, and WIfI classification systems.
3. What information is needed to use the PEDIS classification system for these wounds?
4. Using the information in Chapter 11, what are the factors that might impede this patient's ability to heal?

Doppler Examination

If pulses are faint or not palpable, a hand-held Doppler can detect flow within the vessel; however, the presence of a signal is not an indication of normal flow (**FIGURE 4-17**). Even in the most severe cases of PAD and gangrene, patients may continue to have Doppler signals in the pedal arteries. The Doppler examination is usually begun distally, and is not indicated at the more proximal locations if a pulse is palpable or audible.

FIGURE 4-17

Hand-held Doppler A hand-held Doppler is used to detect blood flow if pulses are not palpable; however, a positive Doppler sound is not indicative of normal blood flow. Doppler sounds can be described as triphasic, biphasic, monophasic, or absent; or they can be described as weak or strong.

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Capillary Refill Time

The severity of microvascular disease (eg, with patients who have diabetes or Raynaud's phenomenon) can be estimated by counting capillary refill time (CRT). CRT is measured by pressing the end of the toe or the skin just proximal to the wound until the color disappears and by measuring the time for recovery to the original color (**FIGURE 4-18**). Normal CRT is less than 3 seconds; however, it is prolonged in extremities with microvascular disease and may vary greatly among patients. Thus CRT is only a screening test and an indicator that more precise vascular testing may be indicated.

FIGURE 4-18

Capillary refill time Capillary refill time is measured by pressing the skin until it blanches, releasing, and counting the seconds it takes to return to normal color. Normal capillary refill time is 2 seconds; more than 3 seconds is indicative of microcirculation or small vessel disease.

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Rubor of Dependency

Rubor of dependency test is a screening procedure for ischemia that can be performed at bedside; thus, it is not definitive but indicative of PAD. The extremity is elevated to 30° and observed for pallor. When the foot with normal circulation is placed into a dependent position, it will become a healthy pink color in approximately 15 seconds. If the reperfusion takes 30 seconds or longer and causes a dark-red or rubor appearance, the test is positive for severe ischemic disease.[16] The faster the pallor appears in the elevated position or the longer it takes for the rubor to appear in the dependent position, the more severe the PAD.

Ankle-Brachial Index

The ankle-brachial index (ABI), a ratio of the ankle systolic blood pressure to the brachial systolic blood pressure, indicates the severity of peripheral arterial disease present in the lower extremity. A 10–12 cm sphygmomanometer cuff is placed just above the ankle and inflated. A hand-held Doppler is

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used to measure the systolic pressure of the posterior tibial and the dorsalis pedis arteries of each leg by noting the pressure at which the pulse returns as the cuff is deflated. Then, the pressures are normalized to the higher of the brachial pressures. The equations for the ABI are as follows:

Right ABI

$$\frac{\text{Higher of the right ankle systolic pressure (posterior tibial or dorsalis pedis)}}{\text{Higher brachial systolic pressure (left or right arm)}}$$Higher of the right ankle systolic pressure(posterior tibial or dorsalis pedis)Higher brachial systolic pressure (left or right arm)

Left ABI

$$\frac{\text{Higher of the left ankle systolic pressure (posterior tibial or dorsalis pedis)}}{\text{Higher brachial systolic pressure (left or right arm)}}$$Higher of the left ankle systolic pressure(posterior tibial or dorsalis pedis)Higher brachial systolic pressure (left or right arm)

The only contraindication for performing the ABI examination is the presence of an ulcer near the ankle, since compression of the ulcer with the sphygmomanometer cuff may cause significant pain to the patient. Interpretations of ABI values are listed in **TABLE 4-4**. In some patients with diabetes, renal insufficiency, or other diseases that cause vascular calcification, the pedal vessels at the ankle become non-compressible. This leads to an elevation of the ankle pressure and consequently an ABI >1.30. In these patients additional non-invasive diagnostic testing is indicated to evaluate the patient for PAD. Another scenario when the ABI may be inaccurate and unreliable is for patients who have heel pressure injuries; the anterior tibial artery may be compressible when the posterior tibial artery is not compressible, making the calculated ABI a misleading indicator of adequate perfusion for wound healing.[17]

TABLE 4-4

Interpretation of Ankle-Brachial Index

1.0–1.2: Normal
0.8–1.0: Minimal peripheral arterial disease. Compression for edema control is safe to use.
0.5–0.8: Moderate peripheral arterial disease, often accompanied by intermittent claudication. Referral to a vascular specialist is advised. Compression therapy is contraindicated if <0.6; modified compression is indicated if 0.6–0.8.
<0.5: Severe ischemia with resting pain. Compression therapy is always contraindicated.
<0.2: Tissue death will occur.
1–1.3: May occur with venous hypertension.
>1.3: Non-reliable in patients with diabetes due to calcification of the arteries.

Ankle-brachial index (ABI) is used to determine the severity of peripheral arterial occlusive disease and to select interventions that will promote healing without causing more tissue necrosis, eg, by using too much compression for lower extremity edema or by debriding a poorly perfused wound with eschar. The test can be easily performed in any clinical setting with a blood pressure cuff and hand-held Doppler.

Toe Pressures

Toe pressures are used in patients with calcified vessels and abnormally high ABI or non-compressible vessels, typical of patients with diabetes. A pneumatic cuff, about 1.2 times the diameter of the digit, is wrapped around the proximal phalanx and a flow sensor is applied distally to the digit. Toe pressures that are greater than 50 are considered normal and those that are less than 50 are abnormal. The majority of patients with foot ulcers or wounds that are caused by PAD have a toe pressure less than 30. In non-diabetic patients, a toe pressure greater than 30 mmHg, and in diabetic patients, 55 mmHg, will usually lead to healing.[18] The normal toe/brachial index, also considered a reliable indicator of lower extremity vascular status, is 0.8–0.99.[19] A toe pressure <30 mmHg or a TBI ≤0.2 infers a diagnosis of severe ischemia or critical limb ischemia.[20]

Segmental Pressures

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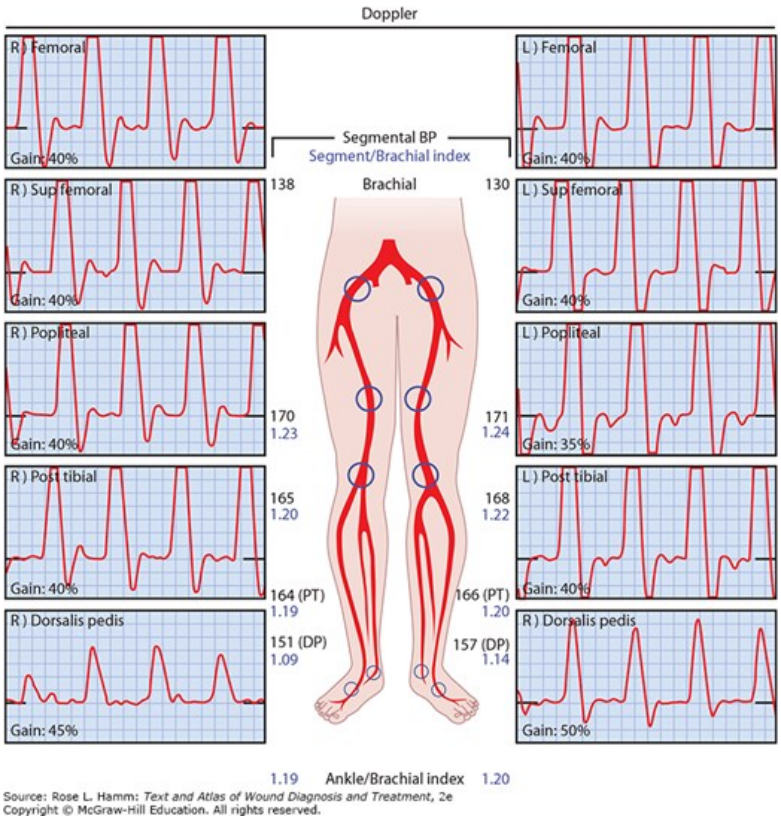
Segmental limb pressure is measured by combining Doppler ultrasound or plethysmography with blood pressure measurements at various locations in the arms and legs. Segmental pressures are a useful adjunct to the physical examination and ABI because they can illustrate differences in blood pressure at specific sites in the extremities and identify gradients that indicate the presence of disease.

A normal variation in the pressures between limb segments is 20–30 mmHg. A difference of 30 or greater indicates significant obstruction. Similarly, variations of pressure between symmetric limb segments do not differ by more than 20 mmHg unless there is an abnormality. Although they are strong indicators, limb pressure measurements are far less sensitive and specific than duplex imaging in the accurate diagnosis of arterial disease (**FIGURE 4-19**).[21]

FIGURE 4-19

Arterial profile includes segmental pressures This study shows arterial waveforms from the femoral artery through the toes. Pressures and waveforms analyses are evaluated at each arterial segment. Triphasic arterial waveforms are identified throughout all segments except the right dorsalis pedis artery where the waveform displays a biphasic pattern. Comparison to the brachial artery pressure allows calculation of the ankle-brachial index.



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Transcutaneous Oxygen Perfusion ($TcPO_2$)

Another important tool in evaluating the patient with the chronic wound is measurement of transcutaneous oxygen measurements ($TcPO_2$), defined as skin oxygenation. The measurement of $TcPO_2$ depends on the cutaneous blood flow, the oxyhemoglobin dissociation, and the diffusion of oxygen through the tissues; thus it reflects the metabolic state of the target tissues. The measurements are taken usually on the dorsum of the foot, antero-medial calf approximately 10 cm below the patella, and in the thigh approximately 10 cm above the patella (**FIGURE 4-20**). The reference site is usually on the chest in the infraclavicular area. The limb is maintained in the dependent position to help increase blood flow.

FIGURE 4-20

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Measuring transcutaneous oxygen tension Transcutaneous oxygen tension, measured on the foot of a patient with arterial wounds on the toes, is a good indicator of healing potential.

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A normal $TcPO_2$ is between 60 and 90 mmHg. A measurement greater than 30 mmHg indicates adequate perfusion for healing, whereas if the tissue surrounding an ulcer has a $TcPO_2$ less than 20 mmHg, the wound typically will not heal. In addition, patients with a $TcPO_2$ less than 20 mmHg will typically experience rest pain. A $TcPO_2$ value greater than 40 mmHg is desirable to support minor amputation site healing (toe amputation or transmetatarsal amputation) (**TABLE 4-5**).

TABLE 4-5

Interpretation of $TcPO_2$

<20 mmHg: Unlikely for healing to occur.
20–30 mmHg: Healing can be expected, but may be delayed.
>30 mmHg: Proximal to the toes: wound can be debrided.
<30 mmHg: Proximal to the toes: wound should not be debrided until revascularization is accomplished.
>40 mmHg: Desired for healing of minor amputation site.
60–90 mmHg: Normal.

Transcutaneous oxygen tension ($TcPO_2$) is a measure of oxygen tension in the skin and is used to determine severity of microvascular disease. The readings also serve as indicators for wound healing potential and safe debridement of necrotic tissue.

Exercise Stress Test

The exercise stress test (EST) assesses PAD in patients with borderline ABIs (0.92–1.0) and symptoms of claudication. A resting ABI is performed first, after which the patient walks on a treadmill (typically at 2 miles per hour with an incline of 10–12%) for 5 minutes or until symptoms of claudication cause the patient to cease the activity. An alternative exercise activity is performance of 50 heel raises in the standing position. The duration of exercise

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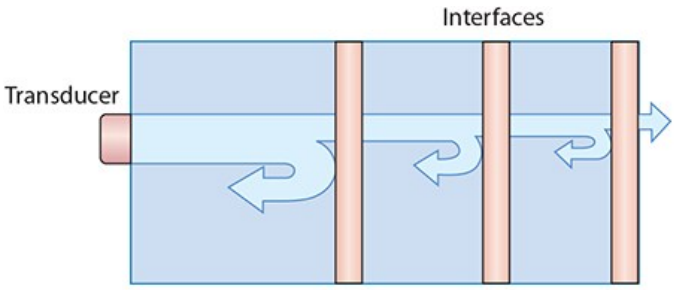
and the nature of the symptoms are recorded. The patient is placed in a supine position and the ankle pressure measurements are recorded every 30 seconds until (1) the BP has returned to baseline or (2) 10 minutes have elapsed. BP values after exercise are compared to pre-exercise values. Normally, there is little or no change in the ankle or brachial BPs; however, PAD sufficient to cause claudication will result in a drop in the ankle pressure of at least 25 mmHg, 25% decline of the resting ankle pressure, or a decrease in the ABI >0.15. A negative EST with the same symptoms suggests cardiorespiratory, neurologic, or musculoskeletal disease.[22]

Arterial Duplex

Ultrasonography is one of the most important diagnostic tools for the vascular system. Many of the terms associated with ultrasound are often used interchangeably and/or incorrectly. A basic understanding of how the technology is applied will help understand when each test is needed.

The principle of ultrasound is bouncing sound waves away from a probe and back to a receiver and measuring the time for the echo or returning sound wave to return after it was emitted. Time and strength of the returned sound waves are amplified and transformed into a visual representation described as A-mode or B-mode. A-mode or "amplitude" mode represents the signal as an amplitude (**FIGURE 4-21**). B-mode or "brightness" mode adds a linear array of scanners that scan a plane through the body to create a 2D image with densities represented as varying degrees of "brightness." This creates a more anatomical representation (**FIGURE 4-22**).

FIGURE 4-21



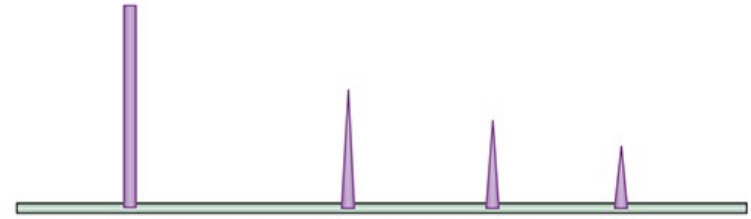


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FIGURE 4-22

Ultrasound, A-mode A-mode ultrasound simply shows the amplitude of the sound waves and is rarely used for diagnostic purposes with the development of Duplex scans. (Used with permission from DeMaria AN, Blanchard DG. Echocardiography. In: Fuster V, Walsh RA, Harrington RA, eds. *Hurst's The Heart*. 13th ed. New York, NY: McGraw-Hill; 2011:chap 18. Available at: http://www.accessmedicine.com.ezproxy.cihe.edu.hk/content.aspx?aID=7805593. Accessed August 23, 2018.)

Ultrasound, B-mode B-mode ultrasound is the reflected sound waves plotted on an oscilloscope to create a two-dimensional image of the anatomical structure. Figure **A** is the external iliac artery without color (the artery is the dark area in the middle of the image), and figure **B** is the right common femoral artery with color added to show direction of flow. Red is typically flow away from the heart and blue is flow toward the heart. A mosaic of color is interpreted as a stenosis, a point at which flow is scattered in both directions.

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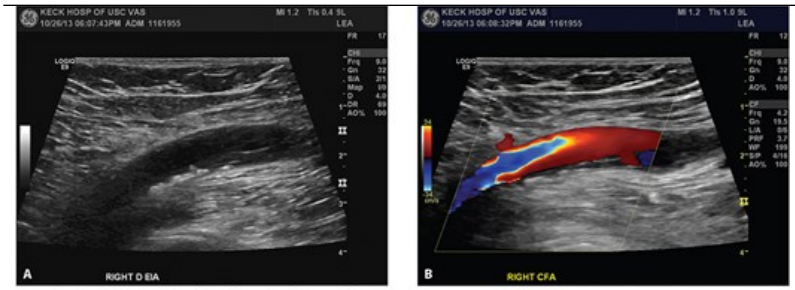

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Doppler ultrasound builds on this by combining the Doppler effect with the ultrasound (**FIGURE 4-23**). Doppler effect is a change in the frequency of a detected wave when the source or the detector is moving, and can measure flow, or in arterial studies, specifically blood flow from the heart to the peripheral extremities. Differences in flow can be represented as changes in color flow Doppler. Additionally, Doppler ultrasound can be continuous or pulsed waves. Pulsed wave Doppler has the advantage of detecting velocities in a small area very accurately, whereas continuous wave can measure higher velocity flows over a larger area. Finally, the term *duplex ultrasound* refers to a simultaneous presentation of Doppler (usually pulsed wave) and 2D or B-mode imaging (**FIGURE 4-24**).

FIGURE 4-23

Doppler with color flow imaging The Doppler effect is used to measure the velocity of blood flow in a vessel and is based upon the principle that flow toward the receiver creates shorter waves than flow away from the receiver. The velocity of flow in the vessel (in this case a vein) is shown on the bottom of the screen in the color flow imaging.

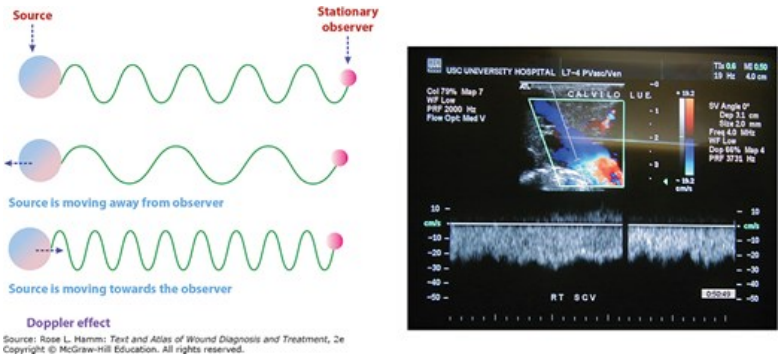

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FIGURE 4-24

Arterial duplex with color flow imaging Arterial duplex scan of the common femoral artery. The flow pattern is laminar in nature (as identified by the homogeneous color of the moving blood through the vessel imaged). The waveform is a triphasic waveform with a peak systolic velocity of 110.9 cm/sec. Signs of a stenotic lesions would show a more mosaic flow color and higher velocities (over 250 cm/sec).

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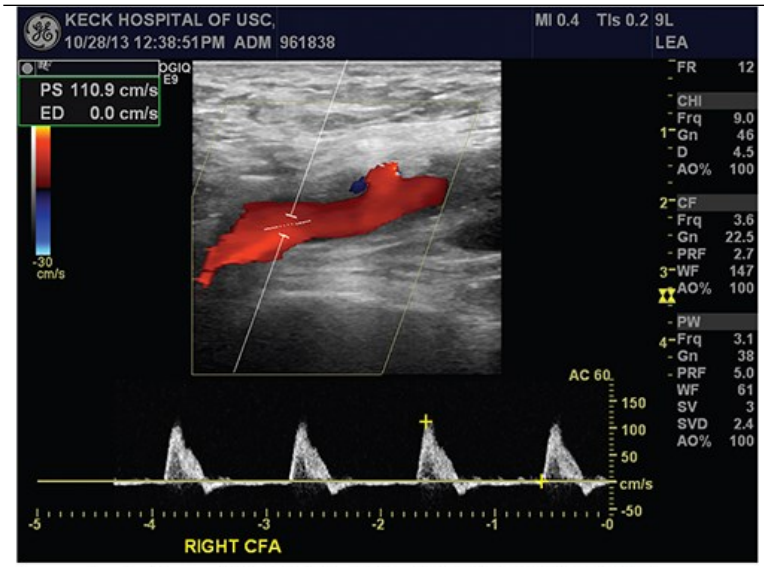

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Duplex ultrasound imaging provides both anatomic and hemodynamic blood flow assessment in a non-invasive manner. Duplex ultrasound can provide spectral analysis, which shows the complete spectrum of frequencies (that is, blood flow velocities) found in the arterial waveform during a single cardiac cycle.

The normal ("triphasic") waveform is made up of three components that correspond to different phases of arterial flow: rapid antegrade flow reaching a peak during systole, transient reversal of flow during early diastole, and slow antegrade flow during late diastole. Lesions are identified by a change in waveform from triphasic to monophasic, or an increase in peak systolic velocity (PSV) followed by a drop in velocity (**FIGURE 4-25A, B**). Certain ratios of the PSV within an area of stenosis to the PSV of the proximal normal segment correlate with the degree of stenosis.

FIGURE 4-25

Arterial waveforms A. Normal arterial waveforms are triphasic and correspond to phases of arterial flow. **B.** Abnormal waveforms occur with macrovascular occlusion and vary from moderate (biphasic) to severe (monophasic and flat), depending on the extent of the occlusion.

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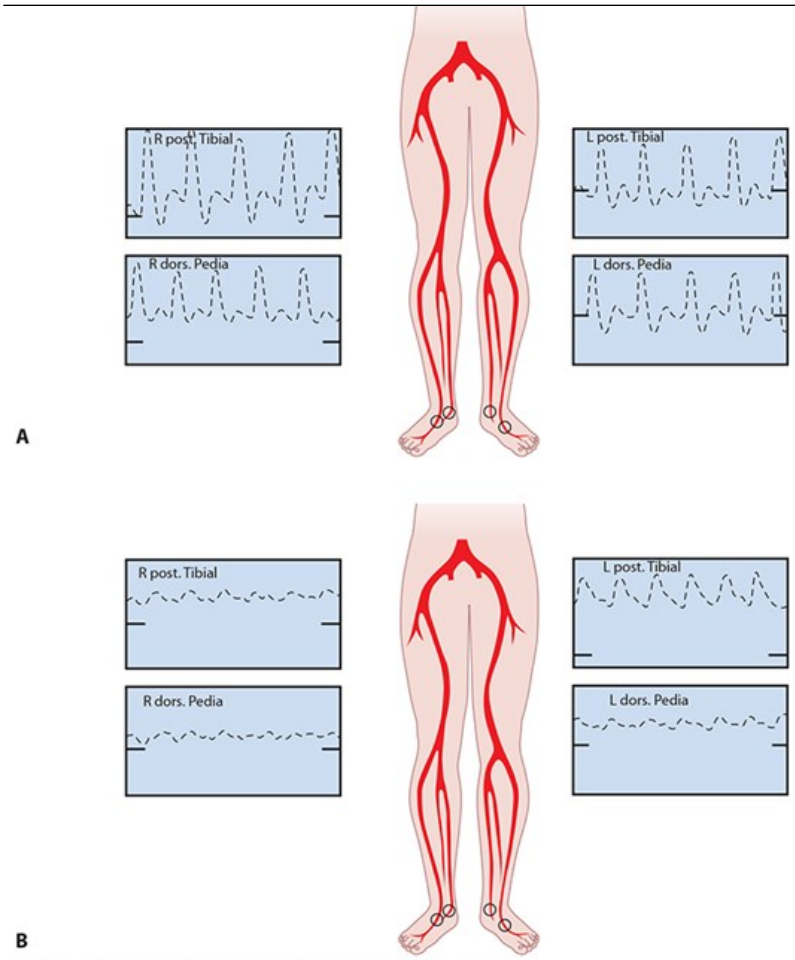

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Computerized Tomographic Arteriography

Accurate evaluation of the arterial blood supply is paramount for successful revascularization of the ischemic extremity for patients with or without diabetes. Arterial imaging is challenging for patients with ischemic complications because they frequently have underlying renal insufficiency, a condition that limits the amount of iodinated contrast that can be injected.

Computerized tomographic arteriography (CTA) involves injection of contrast medium into an upper extremity vein followed by CT scan of the diseased arteries. CTA is a safe, non-invasive procedure that provides high-quality imaging of the arteries; however, significant arterial calcification can impede the transit of contrast in the distal segments of the extremity, in which case more images and higher radiation doses are required. In addition, CTA is only a diagnostic tool and not sufficient for pre-surgical evaluation.

Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) can be performed with a contrast agent injected intravenously, termed *flow dependent*, because the presence of the contrast in the diseased arteries depends upon the blood flow getting to that area. The MRA can also be done without contrast, termed *flow independent*. MRA images are not obscured by arterial calcification; however, the disadvantages are motion artifact, long acquisition times, anxiety in patients who are claustrophobic, and possible nephrogenic systemic fibrosis in the patient with renal insufficiency.

For patients who require revascularization, arteriography using digital subtraction techniques is preferred to determine appropriateness for endovascular revascularization versus surgical bypass (**FIGURES 4-26** and **4-27**).

FIGURE 4-26

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Magnetic resonance arteriography Magnetic resonance arteriography of a male with intermittent claudication. The arrows point to the stenoses in the right common iliac artery and external iliac artery.

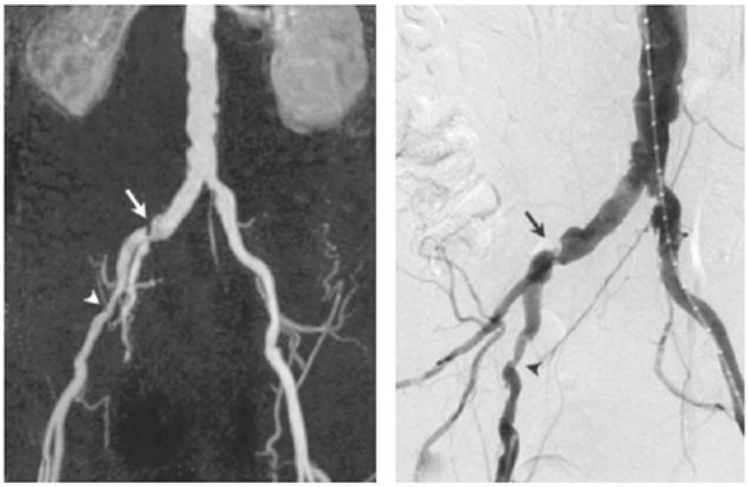

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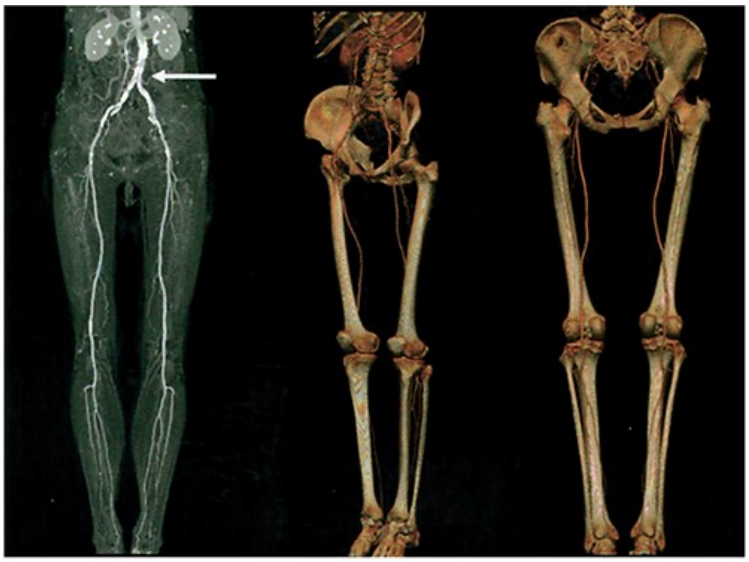
FIGURE 4-27

Computerized tomography arteriography CTA image of a male with calcifications in the abdominal aorta. The peripheral vessels are patent in both lower extremities.



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Invasive Tests

Arteriogram

Angiography (or arteriography when referring specifically to the arterial system) is radiographic imaging of a contrast-filled blood vessel (**FIGURE 4-28**). Despite advances in the quality and availability of the previously described modalities, arteriography remains the gold standard for pre-surgical evaluation of the diseased arteries. There are clinical scenarios in which angiography is not the first choice because of its invasiveness; however, the explosion of endovascular therapy of vascular disease has strengthened angiography as a preferred method because of its sensitivity and ability to be easily combined with endovascular treatment.

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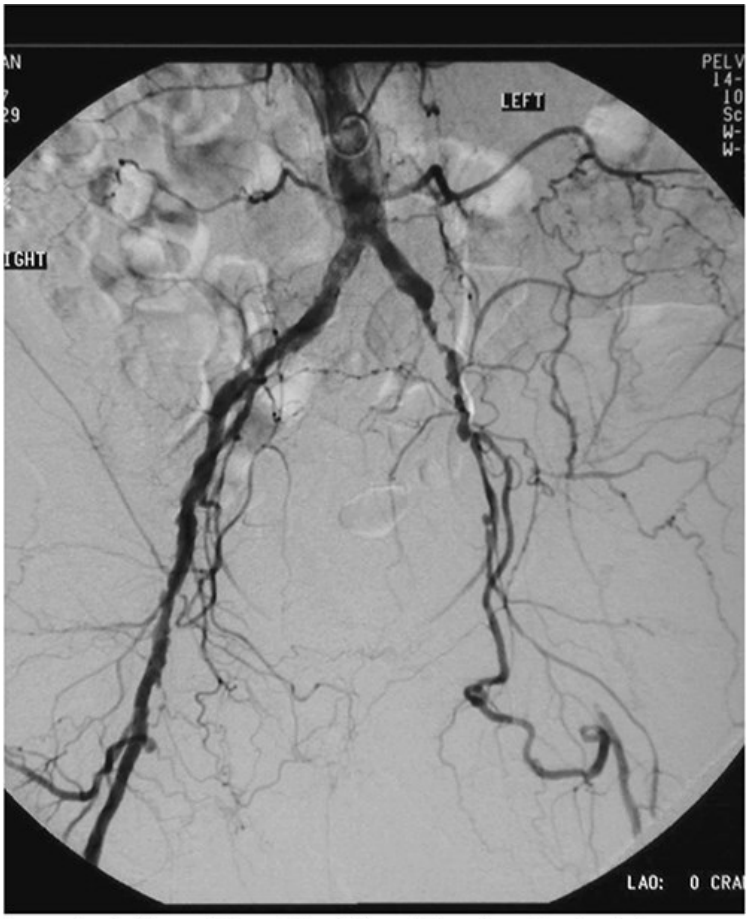
FIGURE 4-28

Arteriogram of lower extremity vessels Arteriogram of the aortoiliac segment of the arterial circulation. Extensive disease is seen in the left distal common iliac and the external iliac is occluded. The vessel seen traversing through the pelvis is the internal iliac artery, which is the main collateral vessel for the occluded external iliac artery on the left.



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Arteriography is performed in a procedure room with a fixed, mounted fluoroscopic unit or in an operating room with a mobile X-ray unit that has its own generator, c-arm, and image intensifier or panel detector. The contrast agent is injected manually or via a power injector into the target vessel. One emerging technique is CO_2 arteriography, in which CO_2 gas is injected to transiently displace blood in the vessel and thereby create an image. CO_2 as a contrast agent is preferable in circumstances where systemic or renal toxicity from iodinated contrast is contraindicated or risky for the patient.

Digital subtraction refers to enhancement of the imaging prior to interpretation. The fluoroscopic image is processed, digitized, and amplified to create a more accurate anatomic picture. Arteriography is done through single-injection with staged or stepped technique (the contrast is injected and images are taken either at various points along the extremity to follow the contrast load or via a rotational method). This technique can create a 3D image of the contrast outline using a detector that rotates between two points as the contrast is injected.

The limitations of arteriography are operator dependence, contrast and radiation exposure, and the inability of the image to effectively characterize the surrounding anatomy or vessel wall.

Prevention

Prevention of arterial wounds is predicated on identifying risk factors and reducing their impact on the integrity of both macro- and microcirculation. Important strategies include the following:

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- Instruct and encourage the patient who smokes to stop by participation in smoking cessation programs.
- Educate the patient about blood glucose controls to minimize the risk of PAD.
- Educate the patient on the need to control hypertension, hyperlipidemia, and hypercholesterolemia through diet and medication.
- Encourage the patient to initiate supervised exercise therapy to develop collateral circulation in the lower extremity, especially in the case of intermittent claudication.[23–26] Exercise tolerance is established by walking on a treadmill until onset of moderate pain and by noting the speed, grade, and time. During treatment, the patient performs the cycle of walking and resting for 30–60 minutes, 3–5 times a week for 6 months. As endurance improves, the speed increases, the grade increases, and the rest time decreases. The goal is not to increase the treatment time, but to increase the activity level, especially the incline grade, within the treatment period.[27] Supervised exercise therapy has been shown to increase walking distance, improve endothelial and mitochondrial function, muscle strength, and endurance, as well as improve overall cardiovascular fitness and quality of life.[24,28]

All patients who have any of the risk factors for atherosclerosis should be medically optimized.[28] Control of hypertension is measured with a goal of systolic pressures less than 140 mmHg and diastolic pressures less than 90 mmHg (for the non-diabetic and non-renal failure patient). Antiplatelet therapy (eg, aspirin or clopidogrel) has been shown to reduce myocardial infarction, stroke, or death in patients with symptomatic lower extremity ischemia. Cholesterol is controlled using statin medications with a goal of less than 100 mg/dL in all patients with PAD. Smoking cessation is critical in halting the progression of atherosclerosis and in reducing bypass graft failure after revascularization.

Treatment

Pre-Surgical Wound Care

Wound care before a patient has revascularization is based on protection of the wounded tissue and at-risk areas (eg, heels and malleoli). Suggested strategies include the following, with the understanding that care is based on the individual patient's presentation, pain, and wound location:

- Debridement of an arterial wound without adequate perfusion for wound healing will make the wound larger. Therefore, intact eschar is not debrided. Infected tissue, termed *wet gangrene*, is debrided either with sharp or surgical techniques.
- The skin and necrotic tissue are kept dry and protected with sterile gauze, lambswool, or cotton between the toes. Swabbing the wound margins with alcohol or iodine solution helps minimize the risk of infection. Dry silver dressings may also be used to prevent maceration and infection while waiting for revascularization and reperfusion (**FIGURE 4-29**).
- The heels are protected from pressure by placing a pillow under the calf and thigh or by using an off-loading boot (**FIGURE 4-30**).
- A foot cradle on the bed helps to keep the weight of bed linens off affected distal digits.
- Post-op shoes help protect the toes from extra pressure, and assistive devices can help further off-load the involved foot.
- In severe cases, the patient is advised to avoid exercise, or limit exercise to functional tasks, in order to decrease the oxygen consumption of surrounding muscles and optimize perfusion to the ischemic areas.
- Elevating the head of the bed 5–7° allows gravity to assist in increasing the blood flow to the foot. Discourage limb elevation that may further reduce blood flow to the distal extremity.
- Keeping the affected extremity warm helps prevent further vasoconstriction due to cold exposure. Hot soaks, hot water bottles, or heating pads are discouraged because the diabetic foot is often insensate and cannot feel burning sensations. Also, because of autonomic neuropathy, the vessels cannot vasodilate to dissipate the heat, resulting in the accumulation of heat in the tissue and subsequent blistering, ulceration, or infection.
- Adjunctive therapies may be indicated for increased microcirculation (eg, electrical stimulation, low-frequency noncontact ultrasound, growth factors, and bioengineered tissue).[29]

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- Cellular and tissue-based products or biological dressings are NOT appropriate for use at this time because the wound environment is not yet optimized.

CASE STUDY

Vascular screening of the patient during the initial evaluation reveals the following:

Left: dorsalis pedis 2+; posterior tibialis 1+ and confirmed by strong Doppler

Right: dorsalis pedis—no palpable pulse, monophasic Doppler; posterior tibialis—no Doppler

Left capillary refill—4 seconds

Right capillary refill—6 seconds

Rubor of dependence is noted on both LEs; patient has positive dependent syndrome on the right.

Temperature of the right forefoot is cool as compared to the left.

The heel wound is 5.5 cm × 3.3 cm; the toe wound is 6.5 cm × 2 cm. There is scant drainage from either wound, no odor, ischemic mottling of the periwound skin, and 8–10/10 pain with any tactile stimulation.

DISCUSSION QUESTIONS

1. What is the appropriate treatment care plan for the patient?
2. What further diagnostic tests are indicated in order to determine the best revascularization technique?
3. Using the UT classification, what would be the type of lesion on both the heel and the forefoot?
4. Is the patient more appropriate for endovascular or open bypass procedure?

FIGURE 4-29

Pre-surgical protection of ischemic digits Lambswool is placed between the toes of an ischemic foot to prevent friction and maceration of the interdigital spaces.

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FIGURE 4-30

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Use of protective boots for the ischemic limb Soft boots provide protection for the heels and certain devices can also increase local tissue perfusion. The heels are floating with no contact with the bed. Non-rigid devices are advised especially if the patient has a plantar flexion contraction.

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Surgical Interventions

If gangrene or *infected tissue* is present on the extremity wound, urgent sharp or surgical debridement of the devitalized tissue is performed. (Refer to Chapter 12, Wound Debridement.) Infection both impedes wound healing and causes pain. If there is inadequate blood flow for healing, debridement of *non-infected tissue* is deferred until after revascularization is performed. After successful revascularization, sharp debridement is performed after signs of angiogenesis (ie, granulation) are visible at the wound edges. If the patient is not a candidate for revascularization, dry eschar should be left intact (**FIGURES 4-31** to **4-33**). Debridement of intact eschar on an extremity with poor healing potential exposes the underlying tissue to possible contamination and infection. Debridement into viable tissue causes further tissue damage, which can lead to extension of the necrosis if severe ischemia is present. The eschar is best kept dry and protected.

FIGURE 4-31

Eschar on ischemic foot Dry attached eschar on an ischemic foot should not be debrided until after revascularization. Removal of necrotic tissue without adequate blood flow results in a larger wound and more tissue loss. The exception is presence of infection that puts the patient or the extremity at risk.

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FIGURE 4-32

Appropriate wound for post-surgical debridement After revascularization, debridement is performed when signs of angiogenesis (ie, granulation at the periphery of the wound) are visible.

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FIGURE 4-33

Should this wound be debrided? Two wounds are visible on this foot, dry eschar on the toes and yellow rubbery eschar at the base of the second toe. Debridement would be determined by patient history of revascularization or by $TcPO_2$ measurements. The dry eschar on the toes would best be left in place and removed as the epithelium migrates under the edges. Enzymatic debridement of the yellow eschar would be indicated, especially if the eschar feels adherent to the underlying tendons.

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Surgical debridement is performed with a scalpel, curette, or hydro-surgery using a device called Versajet (Smith and Nephew, London, UK), or low-frequency contact ultrasound. One method to ensure adequate debridement of devitalized tissue is to "paint" the wound bed or necrotic tissue with methylene blue prior to excision. This gives the clinician an indication of how much tissue needs to be debrided. After the necrotic tissue is removed, principles of moist wound healing are used to facilitate wound closure.

Several adjunct therapies are indicated for debrided wounds, including electrical stimulation, low-frequency non-contact ultrasound, ultraviolet (for infection), pulsed lavage with suction (for removal of coagulum after surgical debridement or minor amputations), and negative pressure wound therapy (NPWT) for wound contraction and stimulation of granulation tissue. The advantages of NPWT include increased granulation and exudate management. Precautions are indicated, however, prior to the formation of collateral circulation and signs of angiogenesis in the wound bed or if hemostasis is not achieved. Use of NPWT in these conditions may cause the wound to deteriorate and form new eschar (**FIGURE 4-34**).

CLINICAL CONSIDERATION

When using NPWT on a debrided arterial wound, reducing the pressure to 40–50 mmHg will prevent compression of superficial capillaries and subsequent tissue necrosis. If the superficial tissue shows signs of necrosis at the time of a dressing change, NPWT should be discontinued.

FIGURE 4-34

Amputation site not appropriate for NPWT Negative pressure on an amputation site before reperfusion occurs can result in necrosis of the wound bed. Likewise, placing negative pressure on the amputation site before hemostasis is obtained will result in the loss of growth factors and cellular components that are needed for wound healing to progress.

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Endovascular Interventions

Advances in endovascular (ie, minimally invasive procedures performed by access through major blood vessels) techniques have made decision making in revascularization increasingly complex. The Trans-Atlantic Inter-Society Document on Management of Peripheral Arterial Disease (TASC) was first written and published in January 2000; it was revised and published in 2007 as TASC II.[11] TASC guidelines categorize aortoiliac and femoropopliteal disease by severity and base intervention recommendations on the extent of the disease.

Options for endovascular therapy are multiple; however, the essential goal is to open the occluded segment with an instrument on a guidewire/catheter as opposed to performing an open incision. Subintimal angioplasty was the earliest endovascular technique in which a wire was used to create a "controlled dissection" at the occluded segment. The wire is traversed through a newly created false lumen and reenters the true lumen past the blockage. Once an opening has been made, the false lumen is expanded.

Another technique is balloon angioplasty by which an expandable balloon mounted on a wire is positioned deflated within the occluded/stenotic segment and then inflated to stretch the lumen to a large diameter. Finally, stents (small expandable tubes) are placed in the occluded segment in order to create an open vessel (**FIGURE 4-35**).

FIGURE 4-35

Endovascular angioplasty for a stenotic artery A catheter is used to pass a closed stent through the artery to the area of stenosis, a balloon is used to expand the stent, and the balloon and catheter are removed. The stent remains in the artery, compressing the plaque against the arterial wall

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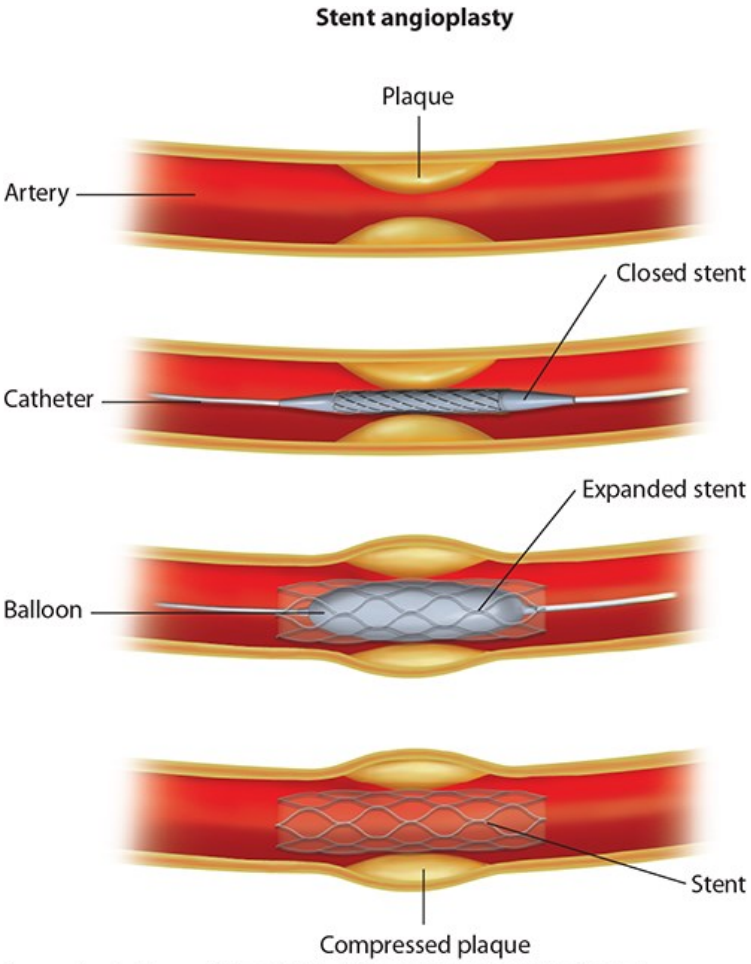
and increasing the size of the arterial lumen. This procedure is used for patients who are not candidates for by-pass surgery and can reperfuse tissue sufficiently for wound healing to progress, albeit slowly.



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Revascularization Surgery

Surgical intervention for PAD is reserved for critical limb ischemia, which is defined as claudication severe enough to interfere with ambulation and daily living, to cause rest pain, or to cause non-healing ischemic wounds. Surgery is dictated by the location and character of the occlusion.

Aortoiliac disease is usually managed with endovascular stenting. For lesions not amenable to endovascular treatment, revascularization is accomplished by a surgical bypass of the occluded segment from healthy proximal vessel (inflow) to healthy distal vessel (outflow) using a prosthetic tube graft. The bypass typically extends from the aorta to the femoral arteries via a transperitoneal or retroperitoneal approach (**FIGURE 4-36**). If the aorta is diseased and is a poor source of inflow, the thoracic aorta, axillary artery, and contralateral femoral artery may be used as the proximal in-flow vessel.

FIGURE 4-36

Illustration of aortofemoral vascular bypass For patients with peripheral vascular disease of the aortoiliac region, a bypass graft from the aorta to both femoral arteries is indicated.

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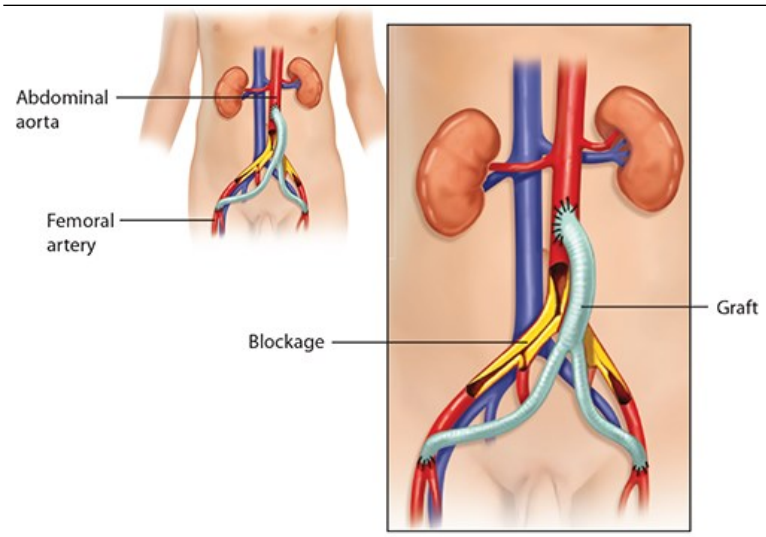

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When disease involves the common femoral and profunda femoral artery, an open groin exploration is indicated, with endarterectomy, profundaplasty, or bypass procedures. The most commonly used graft material (conduit) for bypasses of the lower extremity is the greater saphenous vein (GSV). Polytetrafluoroethylene (PTFE) is also used, with long-term patency rates less than those with GSV.[30,31] Femoropopliteal bypasses are commonly done for occlusions in the SFA and/or popliteal arteries with good distal outflow. Additionally, femoral to infrapopliteal bypasses are performed for lesions of the distal popliteal or proximal tibial arteries.

Post-Surgical Treatment

Post-surgical care of a patient with revascularization focuses on prevention of edema that can lead to dehiscence of the incision, prevention of infection, and meticulous wound care. Edema is managed by elevation, active ankle exercises, and wrapping with short stretch bandages in a spiral technique, especially when the patient begins to ambulate.

Control of medical conditions (eg, diabetes) is an important part of preventing infection, as well as standard precautions on the part of all clinicians caring for the patient. A dry antimicrobial dressing under the wrap may help with high-risk patients, and a recent addition to post-operative care is the disposable negative pressure incisional units that both manage any drainage and prevent shear of the incisional tissue.

Any separation (defined as <1 cm) or dehiscence (defined as >1 cm) of the incision is treated immediately with selective debridement (with meticulous care to avoid the graft), absorbent antimicrobial occlusive dressings, and edema management (**FIGURE 4-37**). After the wound is clean and there is no concern for graft erosion, negative pressure may be used to facilitate wound closure. Using negative pressure can also improve patient function because of the security and occlusivity of the dressings.

FIGURE 4-37

Incisional dehiscence Incisional dehiscence occurs more in patients who are at risk for infection, such as those who have diabetes, protein-energy malnutrition, or obesity.

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The groin incision also needs protection with dry sterile dressings because of the friction and moisture that can accumulate in that area and result in dehiscence.

If the surgery includes a digit, ray, or transmetatarsal amputation, wound care includes the following:

- Removal of any residual coagulum by either soft debridement with sterile gauze or pulsed lavage with suction (**FIGURE 4-38**)
- Selective debridement of devitalized tissue with care to preserve any viable tissue
- Moist wound dressings which may include cellular or tissue products or biological dressings on appropriately debrided, non-infected, and granulating wounds (refer to Chapter 13, Wound Dressings)
- Protective footwear to prevent pressure on the amputation site (**FIGURES 4-39** and **4-40**)
- Gait training with the appropriate assistive device

FIGURE 4-38

Coagulum in a post-amputation site Coagulum in a post-amputation site needs to be removed by pulsed lavage with suction or selective debridement to facilitate wound contraction, decrease the risk of infection, and decrease the amount of non-viable tissue the body has to autolyse. After the wound is clean, negative pressure wound therapy is indicated.

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FIGURE 4-39

Rigid ankle-foot orthotic for the post-surgical ischemic foot Ankle-foot orthoses may be helpful in protecting amputation sites and at-risk heels; however, frequent observation of the Achilles is recommended to ensure pressure relief.

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FIGURE 4-40

Post-operative shoes The shoes can be padded with plastazote or dense foam for the plantar foot and lambswool for the dorsal foot. Early ambulation is an important part of the care plan for any revascularized patient.

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If the amputation is below (BKA) or above (AKA) the knee, application of a stump wrap with short stretch bandages or a stump shrinker is initiated as soon as pain levels will allow. The extremity may also be fitted with a special boot or cast to prevent knee flexion contractures from developing in the early rehab period (**FIGURE 4-41**). When using any type of wrap on an ischemic extremity, daily inspection is recommended to avoid development of soft tissue ischemia or pressure areas on bony prominences.

FIGURE 4-41

Protective boot for below-knee amputation The protective boot for the below-knee amputation provides protection for the incision, maintains knee extension to prevent flexion contractures, provides cushion to prevent pressure points along the limb, and promotes vasodilation for wound healing by keeping the tissue warm.

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Finally, there are some patients with critical limb ischemia and wounds who are not candidates for revascularization or who do not have positive outcomes to procedures. If there are no signs of infection and the edges of the wound remain adhered, conservative care, including meticulous skin care and cleansing of the edges daily with alcohol or iodine solution, can allow the patient to function at a baseline level. Over a period of time, healing can occur by migration of the skin under the eschar. As the edges loosen, they are trimmed with sterile instruments, and with patience and protection, the wounds may heal (**FIGURE 4-42**). Pain management is an integral part of caring for these patients.

FIGURE 4-42

Wound on patient not a candidate for surgery Ischemic wounds that are not or cannot be reperfused with vascular surgery are protected with

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alcohol or betadine cleansing at the edges, dry dressings, and footwear that does not put pressure on the wound.

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Summary

Arterial wounds can be slow and difficult to heal, involving careful management of all aspects of the patient's medical care. Accurate diagnosis of the underlying pathologies and patient-centered decisions on the most appropriate interventions to optimize perfusion are paramount to successful wound healing. Each phase of patient management requires different wound care and changing goals in order to ensure that the patient achieves not just wound closure, but optimal function and quality of life.

VENOUS WOUNDS

Epidemiology

Venous wounds are a consequence of several disease mechanisms, but ultimately result from chronic venous insufficiency (CVI). The reported prevalence of CVI varies from less than 1–40% in females and from less than 1–17% in males.[32] Varying prevalence rates may be due to differences in the classification or definition used, the methods of evaluation to determine wound etiology, and the geographic differences of the regions studied. Some of the better-known risk factors for first-time development of a venous wound include family history, physical activity (typically prolonged standing), history of ankle injury or immobility, and history of deep venous thrombosis.[33] Venous function is also affected by changes in estrogen and progesterone.[34]

CVI is a common illness and its most severe complications, namely venous ulcers, are often debilitating and slow-healing; thus there is a significant economic burden associated with the disorder. An estimated $3 billion is spent annually on the care of venous wounds in the United States.[35]

Anatomy

A review of lower extremity venous anatomy is essential for the understanding of the pathophysiology and treatment of venous disease. The venous system of the leg is comprised of two parallel and connected channels, the deep and superficial systems (**FIGURE 4-1**).

The *superficial* veins of the leg originate at dorsal and deep plantar veins of the foot. The dorsal veins empty into the dorsal venous arch, which is continuous with the great and small saphenous veins. The greater saphenous vein (GSV), which runs anterior to the medial malleolus and medial to the knee, is located in the superficial compartment and connects with the common femoral vein at the saphenofemoral junction (SPJ). Before the SPJ, the GSV receives medial and lateral accessory saphenous veins, as well as tributaries from the groin and anterior abdominal wall.

The small saphenous vein originates from the dorsal venous arch at the lateral foot and runs posterior to the lateral malleolus and into the posterior calf. It then penetrates the superficial fascia of the calf and empties into the popliteal vein.

The *deep* system originates with the digital veins that empty into metatarsal veins comprising the deep venous arch. This continues into the medial and lateral plantar veins, which then empty into the posterior tibial veins. The dorsalis pedis veins on the dorsum of the foot become the anterior tibial veins at the ankle. The paired posterior tibial veins run under the fascia of the deep posterior compartment, join with the paired peroneal and anterior

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tibial veins, and then join the popliteal vein. There are large venous sinuses within the soleus muscle that empty into the posterior tibial and peroneal veins. The popliteal vein enters a window in the adductor magnus, at which point it becomes the femoral vein (formerly superficial femoral vein). The femoral vein receives venous drainage from the profunda femoris vein, or deep femoral vein, after which it becomes the common femoral vein. At the inguinal ligament, the common femoral vein becomes the external iliac vein.

The superficial venous system connects to the deep system via perforating veins that cross through (perforate) the fascial layers. These perforators run perpendicular to the superficial and deep axial veins. The perforators vary in their anatomy and can enter the veins at various points—the foot, medial and lateral calf, and mid- and distal thigh. The valves in the perforator veins aid in preventing reflux from the deep to the superficial system, particularly during periods of standing and ambulation.

CASE STUDY

CONCLUSION

The patient received a right popliteal to posterior tibial bypass graft with the right greater saphenous vein. Four weeks later he had an amputation of the third toe, which had become well-demarcated at the edges with detached eschar, putting the foot at risk for infection. Subsequently, as is sometimes the case, the adjacent second toe became necrotic and required amputation (**FIGURE 4-43**). After removal of both toes, healthy granulation formed and negative pressure wound therapy was initiated on the amputation site (**FIGURE 4-44**).

The heel wound was off-loaded with a foam dressing and protective boots, and the edges were trimmed as the eschar lifted away from new epithelium (**FIGURE 4-45**). When the entire eschar was loose, an escharotomy was performed and the wound treated with collagen matrix and silicone-backed dressings. Both wounds progressed steadily to full closure and the patient was fitted with diabetic shoes and custom-molded inserts (**FIGURE 4-46**).

FIGURE 4-43 A–C.

Post-amputation of the toes

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FIGURE 4-44

Treatment with NPWT

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FIGURE 4-45

Heel wound case study A. The eschar on the patient's heel is becoming detached at the edges, a sign of adequate perfusion for healing. The eschar was trimmed on a weekly basis and was fully debrided when granulation was visible. Healing was slow, but the wound fully closed. **B.** The heel wound after four weeks of debridement, moist wound healing, and off-loading.

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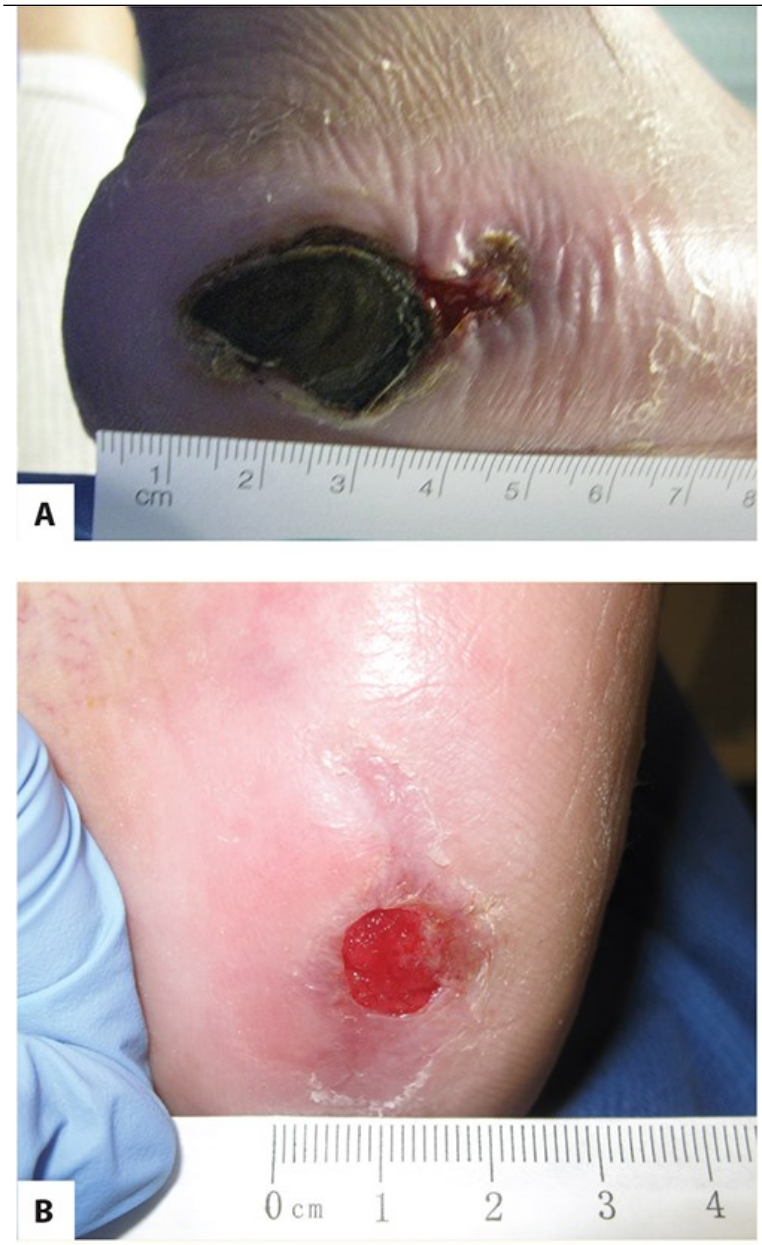

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FIGURE 4-46

Wound almost closed

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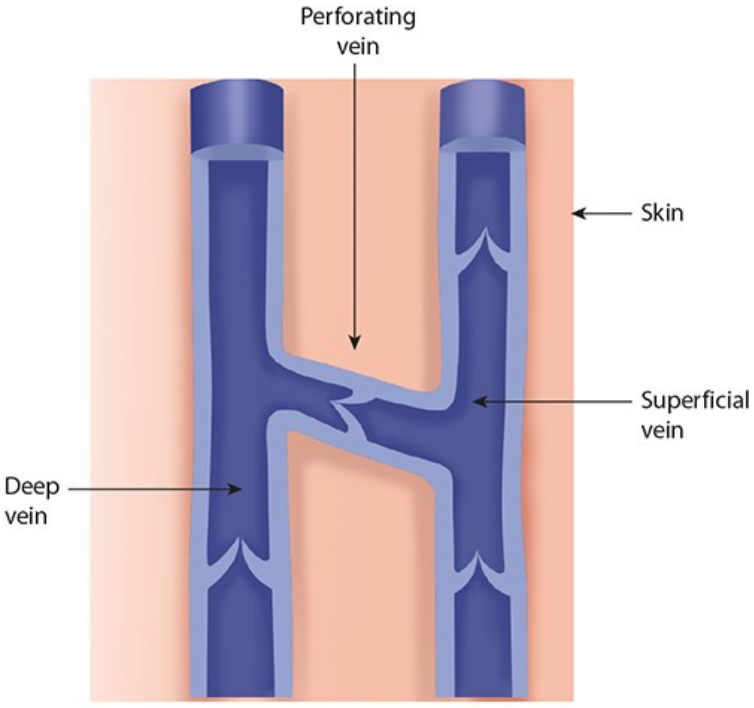
Normal Venous Physiology

The wall of a vein is made up of three layers: the intima, media, and adventitia. Vein walls have less smooth muscle and elastin than arteries do. The adventitia of the venous wall contains adrenergic nerve fibers, especially in cutaneous veins. Sympathetic discharge, thermoregulatory centers in the brain, temperature changes, pain, emotional stimuli, and volume changes can alter venous tone. Venous valves prevent retrograde flow (**FIGURE 4-47**); valvular incompetence, or failure, leads to reflux and its associated symptoms. Venous valves are most prevalent in the distal lower extremity and decrease moving proximally; thus the superior vena cava (SVC) and inferior vena cava (IVC) do not have valves. Because veins do not have significant amounts of elastin, they can withstand large volume shifts with small changes in pressure. This is why most of the capacitance of the vascular tree is in the venous system. The return of the blood to the heart from the leg is facilitated by the muscle pump function of the calf; when the calf muscle compresses the gastrocnemius and soleus sinuses, blood is propelled toward the heart.

FIGURE 4-47

Anatomy of the veins Venous flow is from the interstitial tissue into the venous capillaries, the venules, and the superficial veins, through the perforators into the deep veins. Retrograde flow is prevented by valves located in all three components of the peripheral venous system.



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Etiology

Venous insufficiency is the result of normal venous failure that occurs through several mechanisms that are classified as primary, secondary, or congenital. Ultimately, CVI causes the higher pressure in the deep venous system to be transmitted to the superficial veins (termed *venous hypertension*),[36] resulting in impaired return of venous blood flow to the heart. The underlying pathology is most commonly due to incompetence of the valves of the deep or superficial venous system. (**FIGURE 4-48**). In the scenario of venous valvular incompetence, there is inadequate coaptation of the valves; therefore, the valves do not prevent retrograde flow of blood. Other contributing factors include dysfunction of the gastrocsoleus muscle that works as a venous pump to push blood flow from the superficial veins to the deep veins (**FIGURE 4-49**), inherent vessel factors associated with genetic predisposition, and venous obstruction (ie, deep venous thrombosis or DVT). **TABLE 4-6** lists the risk factors for acquiring venous insufficiency.

FIGURE 4-48

Incompetent valves Normal venous flow is maintained by a series of valves that function like any valve to prevent backwash. With vein distention (eg, with age) the valves do not meet, termed *incompetent valves*, and chronic venous insufficiency results.

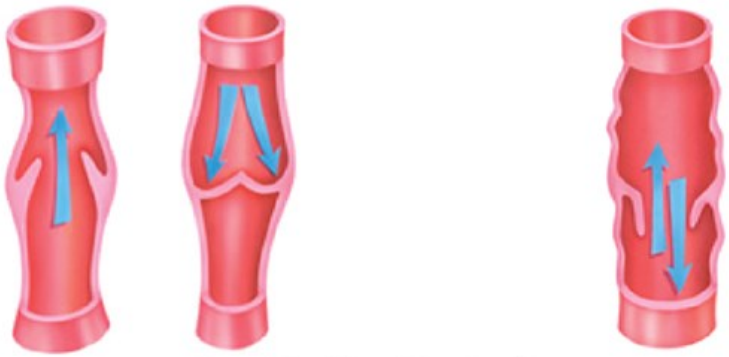

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FIGURE 4-49

Venous pump The gastrocsoleus muscle group works as a venous pump to facilitate the movement of fluid in the distal lower extremity back toward the heart. The pump is activated during any activity that involves gastrocsoleus contraction, especially during ambulation if the ankle range of motion and muscle strength is sufficient to cause compression of the deep veins. The pump action results in compression of the deep vein during toe push-off gait phase, creating a pressure on the vein sufficient to push the blood out of the vein. (**A**) The pressure drops to 15–30 mmHg when the muscle relaxes (during swing, heel strike, and midstance phases of gait). **(B)** This gradient between the deep veins and the superficial veins allows the blood to flow through the perforators and into the deep veins until the next gastrocsoleus contraction, at which point the blood is again forced proximally toward the heart. During the muscle contraction, the proximal valve is open to allow flow of the fluid, and the distal valve is closed to prevent reflux. During the relaxed period, the proximal valve is closed and the distal valve is open, allowing flow through the vein without reflux when the vein is at its largest. If the gastrocsoleus does not function, either from muscle weakness or ankle joint hypomobility, the fluid pools in the deep veins, then in the perforators and superficial veins, and leads to interstitial edema and venous hypertension.

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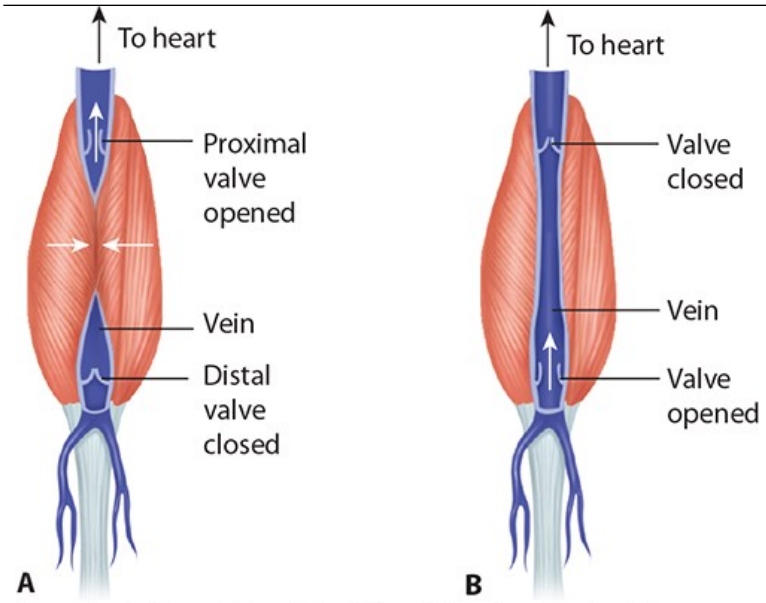

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TABLE 4-6

Risk Factors for Chronic Venous Insufficiency

- Varicose veins
- Deep vein thrombosis
- Previous vein surgery
- Multiple pregnancies
- Congestive heart failure
- Coronary artery bypass surgery with saphenous vein harvesting
- Hip, knee, or ankle trauma
- Ankle immobility
- Prolonged standing
- Family history
- Age over 50
- Obesity

Data from *Association for the Advancement of Wound Care Venous Ulcer Guidelines.* Malvern, PA: Association for the Advancement of Wound Care; December 2010. Available at: http://aawconline.org/professional-resources/resources.

Pathophysiology

In the normal peripheral circulation, blood volume is pumped out of the extremity, and the veins refill from the arterial flow. However, with CVI the veins fill from both arterial flow and retrograde venous flow due to dysfunctional valves, resulting in venous hypertension. Valve failure may be the result of weakness in the vessel wall or valve leaflets (termed *primary*) or may be secondary to injury, superficial phlebitis, or distention caused by hormonal effects or high pressure (termed *secondary*).

The most common manifestation of CVI caused by reflux is varicose veins, or veins that are tortuous and distended (**FIGURE 4-50A, B**). When the incompetent valves are located at junctions of the deep and superficial systems, the high pressure in the deep system is transmitted to the superficial veins. For this reason, varicose veins often start at the saphenofemoral and saphenopopliteal junctions and in the perforating system.

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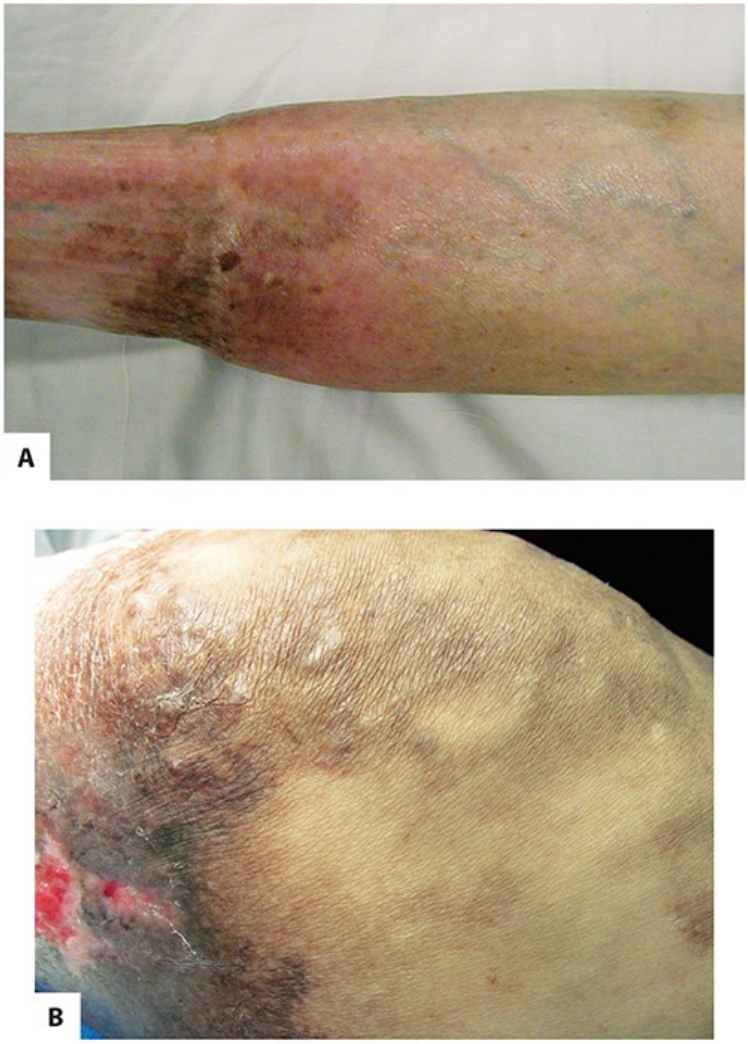
FIGURE 4-50

Varicose veins Varicose veins are enlarged tortuous veins, the result of chronic fluid back-flow from incompetent valves. Varicosities in the superficial veins are visible through the skin. Patient **A** has a healed venous ulcer with hemosiderin staining of the gaiter area and varicose veins in the proximal calf. The constriction seen in the lower leg is the result of socks with elastic at the top, which the patient has been wearing instead of the recommended compression garments. Patient **B** has a congenital condition, Klippel Trenaunay Weber syndrome, with visible varicose veins in the lower thigh.



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Obstruction of the deep veins can also limit the outflow of blood and cause increased venous pressure. The obstruction may occur as a result of an intrinsic process, for example, DVT or venous stenosis. Vascular and integumentary changes that occur after a DVT are termed *post-thrombotic syndrome*. The DVT results in valve destruction that in turn leads to incompetence and persistent venous hypertension both at rest and during ambulation. Signs and symptoms of post-thrombotic syndrome, also termed *post-phlebitic syndrome*, are listed in **TABLE 4-7** and are illustrated in **FIGURE 4-51**. The obstruction can also result from extrinsic compression, for example, in May–Thurner syndrome, a condition in which the right common iliac artery overlies and compresses the left common iliac vein. The compression prevents flow from the left lower extremity and results in higher risk for clotting and edema.

FIGURE 4-51

Post-thrombotic syndrome Patients who have a deep vein thrombosis are at risk for CVI with ulceration. Post-thrombotic syndrome is characterized by the symptoms listed in **Table 4-7** and visualized in the right lower extremity of this patient who had a femoral DVT. Initial symptoms

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included pain distal to the thrombosis, severe edema, and erythema of the lower leg.

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TABLE 4-7

Signs and Symptoms of Post-Thrombotic Syndrome

- Aching or cramping in the extremity
- Feeling that the leg is heavy or tired
- Itching or tingling
- Chronic edema
- Varicose veins
- Hemosiderin staining of the skin
- Ulceration

Post-thrombotic syndrome is a collection of signs and symptoms that occur long-term after a patient has a deep vein thrombosis, which, if untreated, increases a patient's risk of developing a venous wound. Preventive strategies include compression therapy, meticulous skin care, and exercise.

Normal contraction of the calf muscles during ambulation assists in return of blood to the heart by functioning as a mechanical pump (**FIGURE 4-49**). Insufficient contraction of the muscle as a result of muscle weakness or paralysis, joint hypomobility, joint fusion, or gait impairment contributes to ineffective venous emptying and increased venous pressure.

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CLINICAL CONSIDERATION

Evaluating for pitting in the areas with minimal soft tissue (eg, the ankle and gaiter area) will help determine if indeed edema is present. However, edema may be camouflaged in patients with gastrocsoleus atrophy because the leg presents with a fairly normal size as compared to the contralateral extremity.

The ineffective emptying and increased pressure in large vessel hemodynamics are thought to cause venous microangiopathy, defined as derangements in venous function at the cellular level. As venous pressure is elevated, the microscopic anatomy of the vessel breaks down to allow spillage of serum proteins and red blood cells (RBCs) into the interstitial areas where they are trapped by the interstitial edema. When the RBCs die, the hemoglobin is released into the extracellular spaces where it is phagocytosed by macrophages. The by-products of lysed hemoglobin cause the dark, brawny discoloration termed *hemosiderin staining* (**FIGURE 4-52**).[37]

FIGURE 4-52

Hemosiderin deposition in the skin The dark staining in the lower leg of a patient with CVI is a result of the autolysis of entrapped red blood cells and the attached hemoglobin molecules. The by-products of autolysis migrate into the epidermis and produce the discoloration.

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The predominant theories of CVI pathophysiology are (1) fibrin cuff formation, (2) growth factor trapping, and (3) white blood cell trapping. The original theory of the fibrin cuff proposed by Browse and Burnand[38] involves leaking of fibrinogen into the peri-capillary space, thus creating a “cuff” that forms around the venous capillary. This was speculated to increase the diffusion barrier, restrict the diffusion of oxygen into the subcutaneous tissue and skin, and thereby maintain an inflammatory state. This theory has largely been supplanted by other findings but is still thought to play a role in CVI pathology.

The leukocyte-trapping theory involves the sequestering or trapping of leukocytes in the capillaries or post-capillary venules. Venous hypertension results in decreased flow, which in turn causes the accumulation of white cells in the capillaries. The adhesion of activated white blood cells releases inflammatory mediators and proteolytic enzymes that cause endothelial damage. This process may also increase permeability and contribute to protein leak.[39]

Finally, trapping of growth factors by fibrin and other macromolecules is thought to occur via the same mechanism as the trapping of white blood cells. When the growth factors are bound, they are unavailable to facilitate healing.[40]

Clinical Presentation

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As the varicose veins dilate and become tortuous, they may become painful as a result of distention. Patients describe the pain or discomfort of the leg as heaviness, aching, or limb fatigue that is worsened by prolonged standing and relieved by elevation of the extremity. Edema can begin in the foot and ankle and extends up the leg with progressive worsening during the day as the legs are dependent and fluid accumulates. These veins are also prone to superficial thrombophlebitis, as well as occasional bleeding and thinning of the overlying skin.

Skin changes that may also develop include hyperpigmentation in the perimalleolar region from hemosiderin deposition; lipodermatosclerosis with scarring and thickening of the skin secondary to fibrosis in the dermis and subcutaneous fatty tissue; and atrophie blanche, circular whitish and atrophic skin surrounded by dilated capillaries and hyperpigmentation. Brawny edema of the distal calf, "champagne bottle leg," fibrotic and hypertrophic skin, and hyperpigmentation are also visible signs of CVI. These are further described and illustrated in **FIGURES 4-52** to **4-57**. Advanced lipodermatosclerosis may involve fibrosis of the Achilles tendon, thus impairing motor function of the extremity. Patients may also develop eczematous dermatitis, cellulitis, and lymphangitis.

FIGURE 4-53

Lipodermatosclerosis Lipodermatosclerosis, defined as scarring of the skin and fat, is the result of fibrin leaking into the subcutaneous tissue, causing fat necrosis, thickened smooth skin, subcutaneous immotility, and in severe cases, ulceration. Both lower extremities also have extensive hemosiderosis.

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FIGURE 4-54

Atrophie blanche Obstruction of the small vessels to the skin results in atrophie blanche, literally *white skin*, and may be a precursor to ulceration.

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Wounds that have adjacent atrophie blanche will frequently extend into the white area before full healing can occur.

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FIGURE 4-55

Brawny edema Brawny, ordinarily defined as strong and firm, in this case refers to the discoloration from hemosiderin. Brawny edema is characterized by firm, discolored skin with non-pitting edema due to the underlying fibrosis of subcutaneous tissue.

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FIGURE 4-56

Champagne-bottle leg Lipodermatosclerosis of the ankle and gaiter area can prevent flow into that part, resulting in excessive collection of fluid in the calf and the champagne-bottle shape of the leg.

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FIGURE 4-57

Fibrotic, hypertrophic skin In addition to the fibrosis of the subcutaneous tissue, the epidermis can become thick and scaly. If not managed adequately, the skin under the scales can ulcerate or bacteria can collect and cause cellulitis. The erythema seen in this photo is suggestive of cellulitis.

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When evaluating the etiology of edema and ulcerations in CVI, a comprehensive medical and surgical history is crucial, with special attention to family history of varicosities, leg ulceration, thrombotic disorders, previous history of deep venous thrombosis or phlebitis, use of anticoagulation, abnormal clotting factors, unexplained transient unilateral edema, or previous venous-related interventions. Although unilateral edema often suggests venous pathology, a variety of other illnesses with a similar presentation need to be excluded.

Generally, edema that occurs below the knee is due to CVI and edema that extends above the knee to the thigh is due to lymphedema, which is discussed in detail in Chapter 5. Unilateral edema is more likely to be caused by CVI, whereas bilateral edema is usually caused by some systemic disorder (eg, renal failure, kidney failure, congestive heart failure) or by medications. If the edema has a sudden onset, is hard and indurated, and does not respond to compression, the concern is obstruction of the pelvic or abdominal lymph nodes, which is most commonly from a malignancy (**FIGURE 4-58**). The correct differential diagnosis is imperative for effective interventions.

FIGURE 4-58

Lower extremity edema due to lymph node obstruction Sudden onset of hard indurating edema that does not respond to compression is suggestive of abdominal or groin lymph node obstruction, frequently related to metastatic disease. Urgent medical referral is imperative for timely diagnosis and treatment.

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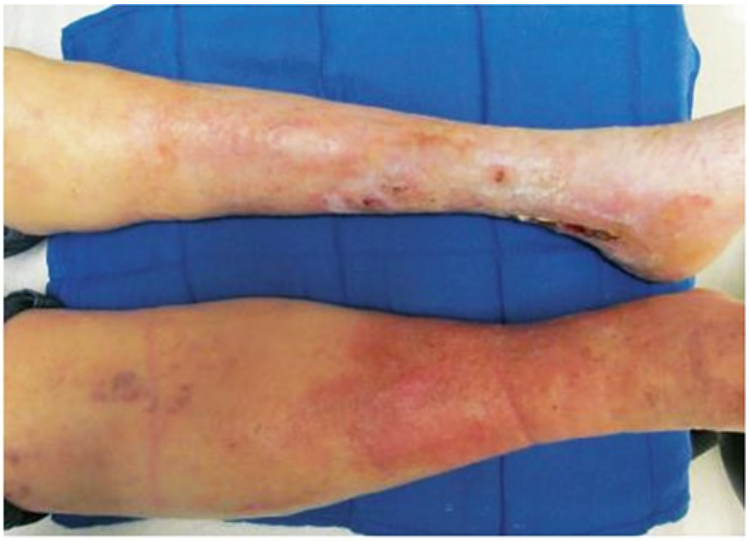

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Additional signs and symptoms that are typical of venous wounds include the following:

- *Location:* Venous wounds occur above the medial and lateral malleoli in the distal third of the lower leg, termed the *gaiter area* (**FIGURE 4-59**). If the wound is outside of this area (eg, directly on the malleolus, on the post-malleolar skin, on the calf, or on the proximal leg), the origin is probably not venous.
- *History of wound formation:* Venous wounds tend to be preceded and surrounded by changes in color and texture of the skin and have a more insidious development. If there are no skin changes and the wound onset was sudden, even in the presence of edema, other causes need to be considered. For example, a traumatic wound, a wound secondary to sickle cell disease, or a surgical incision may cause edema that then impedes wound healing; however, it would not be diagnosed as a venous wound.
- *Wound appearance:* Venous wounds tend to have uneven edges, shallow depth, and a fibrotic or granular wound base (**FIGURE 4-60**). If the wound has been present for a long period, the edges may be senescent or rolled. There is usually little if any eschar.
- *Periwound skin:* Changes in the periwound skin include increased dermal thickness (eg, >1.985 mm measured with high frequency ultrasound), hemosiderin staining, crusting or scaling, lipodermatosclerosis, varicosities, or atrophie blanche.[41]
- *Drainage:* Just as water flows from a hole in a hose or water pipe, fluid flows from a wound on an edematous extremity. Drainage from a venous wound is usually serous; however, if there is infection present, it may be thick, purulent, and odiferous. If a wound appears venous in all respects but tends to have little or no drainage, there is concern for arterial insufficiency as well.
- *Pain:* Venous wounds are not typically painful like ischemic wounds. Wounds that are exquisitely painful to any tactile stimulation are more likely to be vasculitic or arterial (**FIGURE 4-61**).

Skin temperature elevated more than 1.1°C or 3°F is suggestive of infection, along with erythema of the periwound skin, increased drainage with odor, friable granulation tissue, and pain.[42] Wounds that fail to improve within 6 weeks after initiation of treatment are very likely not a result of CVI and should be investigated for other diagnoses (**TABLE 4-8**). Studies have also indicated that wounds that decrease by 40% in the first 3 weeks of treatment have higher healing rate.[37]

FIGURE 4-59

Location of a venous wound Venous wounds are located in the medial or lateral gaiter area, defined as the distal third of the lower leg. Wounds outside this area, including over or distal to the ankle, on the calf, or on the anterior shin, are usually caused by some other etiology although chronic edema may impede the healing.

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FIGURE 4-60

Typical appearance of a venous wound A venous wound typically has serpentine, or uneven, edges; striated granulation or yellow fibrous tissue; serous or serosanguineous drainage; and rolled edges. Wounds with even, punched-out edges should be evaluated for an arterial component.

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FIGURE 4-61

Vasculitic wounds on patient with systemic lupus erythematosus Vasculitic wounds may be close to the typical location of venous wounds, but as in this photo, can be anywhere on the extremity. If they appear on or distal to the ankle, the wounds are probably not venous in origin, although the vasculitic inflammation may cause edema. In addition, vasculitic wounds are very painful, are usually smaller than most venous wounds, and require treatment of the underlying vasculitis in addition to wound care.

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TABLE 4-8

Differential Diagnosis of Venous Wounds

Wounds that fail to heal after 6 weeks of evidence-based care (compression, moist wound healing, exercise) should be studied further for diagnoses that may appear as venous wounds but are indeed caused by other pathologies[37]:

- Malignancies
- Vasculitis
- Polyarteritis nodosum
- Vasculopathy
- Pyoderma gangrenosum
- Mycobacterial or fungal infections
- Necrobiosis lipoidica diabeticorum
- Systemic lupus erythematosus wounds
- Cryoglobulinemia

Classification of Venous Wounds

The International Consensus Committee on Chronic Venous Disease has developed a classification of chronic venous insufficiency, designed to standardize parameters for medical and surgical research. In the CEAP classification, each letter stands for a particular dimension of the disease: ***C*** represents the clinical signs, ***E*** stands for the etiology, ***A*** is for the anatomy involved, and ***P*** is the pathophysiology.[43–45] Refer to **TABLE 4-9** for a complete outline of the CEAP classification with illustrations.

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CASE STUDY

Mrs. VB is a 52-year-old female who has a history of non-healing wounds on the right medial and lateral lower extremity in the gaiter area, of more than 1-year duration (**FIGURES 4-62** and **4-63**).

SUBJECTIVE INFORMATION

- Patient works as a manager for a department store and wants to continue to work while she is being treated.
- Pain levels are 6–8/10 almost constantly and 10/10 with any tactile stimulation to the wound bed.
- Patient is currently applying Silvadene to the wounds daily and covers them with cotton gauze and pads, anchored with tape.
- Patient lives in a one-story home with her husband and two dogs.
- Patient reports copious drainage that interferes with her social and professional life, as well as keeping her awake at night.

MEDICAL HISTORY

- History of hypertension
- History of hysterectomy about 5 years previously; no pregnancies reported
- History of obesity
- History of deep venous thrombosis in the right femoral vein
- Negative for diabetes, cardiac disease, and peripheral arterial disease

VASCULAR EXAMINATION

- Pulses—right posterior tibial not palpable because of the wound, strong Doppler signal; right dorsalis pedis 2+; left pedal pulses 3+

DISCUSSION QUESTIONS

1. What skin changes are observed in the lower extremity?
2. Describe the tissue in the wound bed.
3. What other information is needed to determine the cause of the patient's wounds?
4. Classify the patient's venous disease and wounds using the CEAP system.
5. Which non-invasive tests are indicated for this patient and why? What would you expect to learn about the patient in order to make a diagnosis?
6. Which invasive tests are indicated and why?

FIGURE 4-62

Right lower extremity of case study

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FIGURE 4-63

Left lower extremity of case study

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TABLE 4-9

CEAP Classification of Venous Wounds

C represents the clinical signs, **E** stands for the etiology, **A** is for the	6 = Skin changes as defined above with active ulceration

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anatomy involved, and **P** is the pathophysiology.

Clinical signs are identified from the following list, then designated symptomatic or asymptomatic:

0 = No visible or palpable signs of venous disease

1 = Telangiectasia or reticular veins—small dilated veins (0.5–1 mm in diameter) that develop in the superficial skin as a result of venous hypertension

2 = Varicose veins—dilated, tortuous veins in the lower leg that signify the progression of CVI due to venous hypertension

3 = Edema without ulceration

S: symptomatic, including ache, pain, tightness, skin irritation, heaviness, and muscle cramps, and other complaints attributable to venous dysfunction

A: asymptomatic

Etiology is defined as one of the following:

- Ec: Congenital (for example, congenital valvular dysfunction)
- Ep: Primary, in which venous insufficiency is the primary disease process observed
- Es: Secondary, in which another disease process occurring first (for example, acquired lymphatic insufficiency)
- En: No venous cause identified

Anatomical class is divided into the following:

- As: Superficial veins
- Ad: Deep veins
- Ap: Perforating veins
- An: No venous location identified

Superficial veins:

1. Telangiectasia or reticular veins
2. Long saphenous vein above the knee
3. Long saphenous vein below the knee
4. Short saphenous vein
5. Nonsaphenous system

Deep veins:

6. Inferior vena cava
7. Common iliac vein
8. Internal iliac vein
9. External iliac vein
10. Pelvic, gonadal, broad ligament, other veins
11. Common femoral vein
12. Deep femoral vein
13. Superficial femoral vein
14. Popliteal vein
15. Crural, anterior tibial, posterior tibial, peroneal veins

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4 = Skin changes ascribed to venous disease (pigmentation, venous eczema, lipodermatosclerosis)

5 = Skin changes as defined above with healed ulceration

16. Muscular, gastrocnemial, soleal veins

Perforating veins:

17. Thigh
18. Calf

Pathophysiology is classified as follows:

Pr: Reflux, usually from incompetent valves

Po: Obstruction, usually from DVT; refers to a total occlusion of one of the veins at any point *or* more than 50% narrowing of at least half of a vein segment

Pb: Both

Pn: No venous pathophysiology identifiable

Data from Porter JM, Moneta GL. Reporting standards in venous disease: an update. International Consensus Committee on Chronic Venous Disease. *J Vasc Surg.* 1995;21:635–645. Eklof B, Rutherford RB, Bergan JJ, et al. Revision of the CEAP classification for chronic venous disorders: consensus statement. *J Vasc Surg.* 2004;40:1248–1252. Meissner MH, Gloviczki P, Bergan J, et al. Primary chronic venous disorders. *J Vasc Surg.* 2007;46:54S–67S.

With the CEAP classification in mind, there are four questions to be addressed during the process of evaluating a patient with a venous wound.

1. What are the clinical signs present in the lower extremity?
2. What is the etiology of the venous stasis disease?
3. Where is the anatomic disease process occurring?
4. Is the pathology at that anatomical location obstruction or incompetent valves resulting in reflux, or a combination of the two?

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In addition, the clinician needs to identify other disease processes, confirm infection, and identify contributing factors that can be addressed in the care plan.

Patient Examination

A comprehensive examination of any patient with a wound below the knee, including a venous wound, begins with an assessment of the arterial circulation, as discussed in the section on arterial wounds. A notable sign that arterial insufficiency may be impeding the wound healing is lack of hair growth in the lower half of the leg and foot.[37] An ankle-brachial index (a ratio of the blood pressure measured at the ankle and arm) is indicated if there is any sign of diminished flow, and results less than 0.8 suggest that the patient should be referred to a vascular specialist.[46]

The venous system is assessed with the patient in both standing and supine positions. Standing increases venous hypertension and dilates the veins, thereby facilitating the examination. Patients with superficial valvular incompetence commonly exhibit palpable great saphenous veins. Palpable cords may also be present (**FIGURE 4-64**). CVI dermatitis at the distal ankle can mimic eczema or dermatitis of another cause. For this reason, history and physical findings must be weighed alongside focused vascular studies, both invasive and noninvasive.

FIGURE 4-64

Distended saphenous vein Distended veins are visible in the lower leg as a result of superficial valvular incompetence or with compression of the blood pressure cuff around the calf when testing for augmented venous flow.

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A thorough wound assessment includes all of the components discussed in Chapter 3. The clinical findings are used to select interventions, determine outcomes, and measure progress.

Non-invasive Vascular Studies

Venous Duplex Examination

Duplex ultrasound for superficial/deep/perforating vein assessment has largely replaced the Brodie–Trendelenburg and Perthes tests. There are different types of venous ultrasonography, including compression ultrasound (B-mode imaging only), duplex ultrasound (B-mode imaging and Doppler waveform analysis), and color Doppler imaging. Different lower extremity veins are best evaluated with different techniques. Compression ultrasound is typically performed on the proximal deep veins (the common femoral, femoral, and popliteal veins), whereas a combination of duplex

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ultrasound and color Doppler imaging is used to evaluate the calf and iliac veins.[47]

Valve reflux is identified with distal augmentation of flow and release, normal deep breathing, and performance of a Valsalva maneuver. Augmentation of flow is achieved by compressing the leg distal to the ultrasound probe in order to exaggerate the natural calf-pump mechanism and increase fluid return. With the patient standing, the probe is used to obtain sample volumes from the femoral and saphenous veins. Sudden release of augmentation allows the assessment of reflux and valvular competence. The small saphenous vein and popliteal veins are then examined. Perforator veins can also be assessed this way.[42]

In addition, assessment of microcirculation by transcutaneous oxygen tension can predict wound healing potential as discussed in the section on arterial wounds. Values of more than 30 mmHg in the periwound skin suggest sufficient oxygenation for healing to occur.

Invasive Testing

Invasive vascular studies include phlebography or venography, ambulatory venous pressure, and intravenous ultrasound. Phlebography uses intravenous contrast and radiography to help distinguish saphenofemoral anatomy and reflux, and thus primary from secondary venous insufficiency. Although its routine use has largely been replaced by venous duplex imaging, phlebography is occasionally performed before deep vein reconstruction or in patients with inconclusive duplex results before other venous surgery.

CLINICAL CONSIDERATION

Approximation of central venous pressure is a screening test to determine if an element of congestive heart failure (CHF) is contributing to lower extremity edema. The hand is held below the heart until the veins are visibly filled. When the hand is elevated slightly above heart level, the veins flatten if there is minimal risk of CHF being present because the fluid can return to the central vascular system. If, however, the veins remain distended, referral to the physician for further medical care is advised before any compression therapy is initiated. Prominent jugular venous distention is another visible sign of increased central venous pressure with CHF.

Ambulatory venous pressure is considered the hemodynamic gold standard for the assessment of CVI[48]; however, it is seldom used in clinical practice because of its invasive nature, concern about its accuracy, and availability of numerous alternative diagnostic modalities. A needle is inserted into the pedal vein and connected to a pressure transducer; pressure is measured at rest and after exercise, as well as before and after placement of an ankle cuff to distinguish deep from superficial venous disease.

Intravascular ultrasound has gained acceptance in the management of venous disease and is increasingly being used to help guide interventions. A catheter-based ultrasound probe is used to visualize periluminal vessel anatomy in order to assess for obstructive disease. Intravascular ultrasound may be superior to venography in estimating the morphology and severity of central venous stenosis, especially in the pelvis, and in visualizing the details of intraluminal anatomy.[49]

Prevention

Venous ulcer guideline documents based on extensive database searches to review reliable research provide evidence-based standards of care for patients with venous disease. One document is from the Association for the Advancement of Wound Care, which uses the same level of evidence criteria that the National Pressure Ulcer Advisory Panel uses to evaluate interventions for pressure ulcers. (See Chapter 6, Pressure Ulcers/Injuries.) The Wound Healing Society used a similar method of evaluating the data, but included animal studies to support suggested strategies.[50,51] The guidelines published by these two organizations dedicated to providing the best evidence-based care to all patients with wounds are the basis of this discussion on prevention and treatment.

Prevention is predicated on first determining the risk factors of any individual, then providing the education needed to effect lifestyle changes, including causes of skin breakdown, principles of good skin care, smoking cessation, how and why to use compression, and appropriate exercise (**TABLE 4-10**).[37] Additional strategies are to avoid prolonged standing and sitting, avoid crossing the legs, and wear light compression garments, especially for flying and for prolonged standing or sitting. Elevation with the leg higher than the heart is supported for prevention and treatment;

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however, it is not sufficient in and of itself. Elevation is only a principle to use when sitting or supine, and is not a substitute for compression and exercise.

CLINICAL CONSIDERATION

An engaging way for a patient to perform ankle exercises is to "write" the alphabet with the big toe while sitting on the edge of a chair. This activates the lower leg muscles in all the compartments and mobilizes the ankle joint in all directions.

TABLE 4-10

Exercises for the Patient with Venous Wounds

- Gastrocsoleus stretches to optimize ankle range of motion
- Ankle pumps and circumduction
- Heel/toe raises in both sitting and standing positions
- Ankle rocker board exercises
- Step over a 3- to 4-inch obstacle using a heel strike in front, toe push-off in back
- Exaggerated heel/toe sequence during ambulation
- Walking or bicycling for fun

Non-Surgical Treatment

Selection of treatment strategies is guided by the disease severity using the CEAP classification system in a stepwise fashion. The mainstay of initial treatment for CVI is non-surgical measures to reduce symptoms, prevent the development of secondary complications, and halt disease progression. The four basic components of wound treatment include compression, treatment of infection, moist wound healing, and exercise to activate and strengthen the venous pump.

Compression

Two questions drive the selection of compression for the patient with a venous wound: (1) What needs to be wrapped? and (2) What materials are best for the patient?

An algorithm for the first question is presented in **FIGURE 4-65**. Bilateral edema *can* be present as a result of reflux or obstruction; however, systemic diseases that cause edema need to be ruled out and treated. In some cases of CHF, compression is deferred until the patient is stabilized and there is no concern about overloading the heart with fluid from the extremities.

FIGURE 4-65

Algorithm for deciding *what* to wrap when treating a patient with a venous wound Compression selection begins with a careful assessment of the edema. If the patient has bilateral lower extremity edema, systemic disorders such as congestive heart failure, kidney failure, or liver disease must be ruled out, as well as carefully reviewing the medications. (See Chapter 5, Lymphedema.) If the patient has acute congestive heart failure, compression may need to be deferred until the patient is diuresed and there is no risk of overloading the heart. Any systemic issue must be addressed in order for local treatment to be effective. If the edema extends above the knee, there is probably secondary lymphedema, which would be treated with manual lymphatic drainage, exercise, and compression applied from toe to upper thigh. If the edema is limited to below the knee, the next step is to evaluate the vascular status to determine the type of material that is best for the individual patient (Used with permission from Rose Hamm.)

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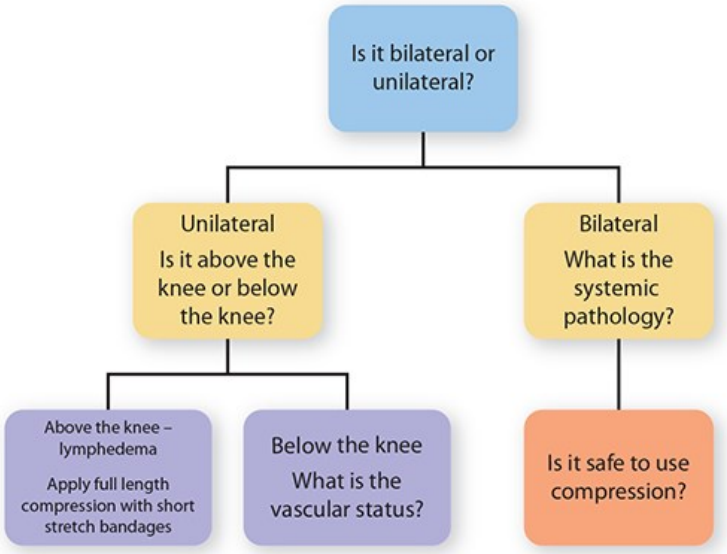
Is it bilateral or unilateral?
Unilateral
Is it above the knee or below the knee?
Bilateral
What is the systemic pathology?
Above the knee – lymphedema
Apply full length compression with short stretch bandages
Below the knee
What is the vascular status?
Is it safe to use compression?

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An algorithm for selection of materials and wrapping technique based on the lower extremity ABI is presented in **FIGURE 4-66**. Materials are classified as elastic, non-elastic, rigid, stiff, single-layer, and multilayer, and wrapping can be performed in a spiral or a figure-8 pattern. The principles of compression are based on the following: (1) there must be a gradient between the pressure at the ankle and at the calf in order to push the fluid cephalad, and the usual differential for a limb with no arterial disease is 40 mmHg at the ankle and 18 mmHg at the calf,[52] and (2) Laplace's law, which has been modified to determine the sub-bandage pressure by using the following equation:

$\text{Pressure} = \frac{T \times N \times 4630}{C \times W}$ Pressure = T × N × 4630C × W

Pressure = the sub-bandage pressure, also termed the *compression force*

T = the tension of the material, determined by the material and how much it is stretched

N = the number of layers of material used

4630 = a constant calculated using the original LaPlace's Law

C = circumference of the extremity being compressed

W = width of the bandage

FIGURE 4-66

Algorithm for selection of appropriate compression therapy Selection of the appropriate compression therapy is based on vascular examination and patient comfort and tolerance. Every garment, compression system, elastic or non-elastic wrap has a tension that determines the amount of pressure when properly applied. Manufacturer's guidelines should be followed carefully to avoid complications that can occur with inappropriate compression therapy. Compression of any type is generally contraindicated if the ABI is less than 0.5; however, if the patient can tolerate multiple layers of soft gauze wrapping, it may be beneficial in activating the lymphatics as well as anchoring the appropriate primary dressing. (Used with permission from Rose Hamm.)

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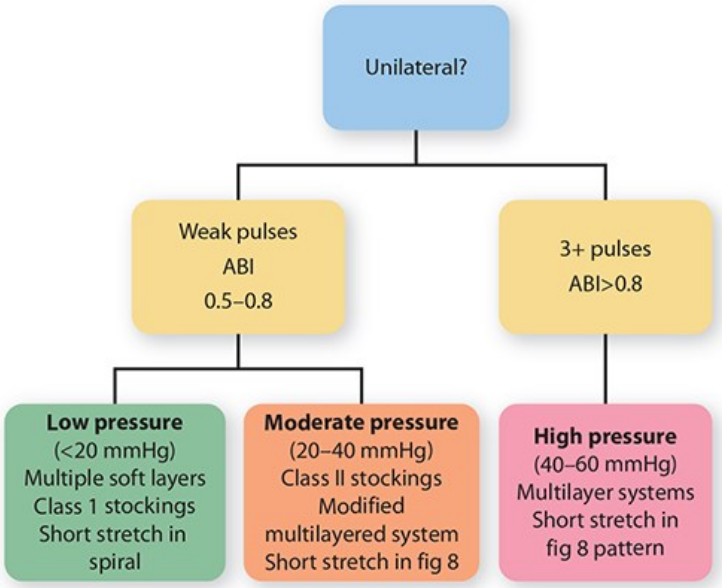

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Using the principles in the equation, the sub-bandage pressure can be altered to meet the needs of an individual by changing any of the components of the equations. Some of the more common applications of these principles in the clinical setting include the following:

- Tension is varied by the kind of material used and how much it is stretched. Stiffer materials provide more pressure, and elastic materials provide more compression as they are stretched. Usually appropriate pressure with elastic materials is calculated on a 50% stretch (eg, the elastic component of a multilayer compression system). Consistent tension on any material used is needed for consistency and to avoid high/low pressure areas along the extremity.
- The number of layers provided by a wrap depends upon the technique used (eg, a spiral wrap with a 50% overlap will result in 2 layers; a spiral wrap with 66% overlap, 3 layers; and figure-8, 4 layers). If all other factors are equal, a figure-8 wrap will produce two times the compression force of a spiral wrap (**FIGURES 4-67** and **4-68**).
- If the extremity has no contour, the compression will be the same throughout the entire length, if the other factors are equivalent. In order to obtain a pressure gradient between the ankle and calf, there must be a circumference differential. If the limb is shaped like a pencil (ie, with no difference in size between the ankle and the calf), extra padding can be placed around the calf to create a cone shape that results in the ankle/calf differential. Also, if the limb is shaped like a champagne bottle, padding can be placed around the ankle to even out the shape and reduce the gradient to 40/18 mmHg (**FIGURES 4-69** and **4-70**).
- Wide bandages provide less compression than narrow ones; thus using narrow bandages distally can help create greater pressure around the ankle, and using wider bandages around the calf can help reduce the proximal pressure. This principle is used especially when wrapping toe to thigh for lymphedema. In addition, narrow bandages are easier to apply around the ankle without creating wrinkles that can cause high-pressure spots.

CLINICAL CONSIDERATION

While LaPlace's Law supplies the science of compression, the art is in the hands of the clinician who is applying the bandages. Absolute and consistent tension on the bandages, meticulous placement for appropriate overlap, and avoidance of wrinkles are necessary for patient comfort and optimal outcomes.

FIGURE 4-67

Spiral wrap of the lower extremity A spiral wrap is performed with the bandage kept at a 30–45 degree angle and can have a 50% overlap (providing two layers of compression) or 66% overlap (providing three layers of compression). Consistency of overlap and tension is essential for

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optimal therapy and requires practice, practice, and more practice for any clinician.

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FIGURE 4-68

Figure-8 wrap A. Compression of the lower extremity must include the foot and ankle. Two or three wraps around the foot and figure-8 around the ankle are the best techniques to anchor the bandages and prevent wrinkles that can abrade and blister the skin. Tape over the "seams" of the wrap prevent loosening and slippage of the bandages. **B.** Figure-8 wrap provides four layers of compression and is performed with consistent 50% overlap and a 45-degree angle of the bandages. A consistent chevron appearance on the anterior leg provides feedback to the clinician about the quality of the wrap.

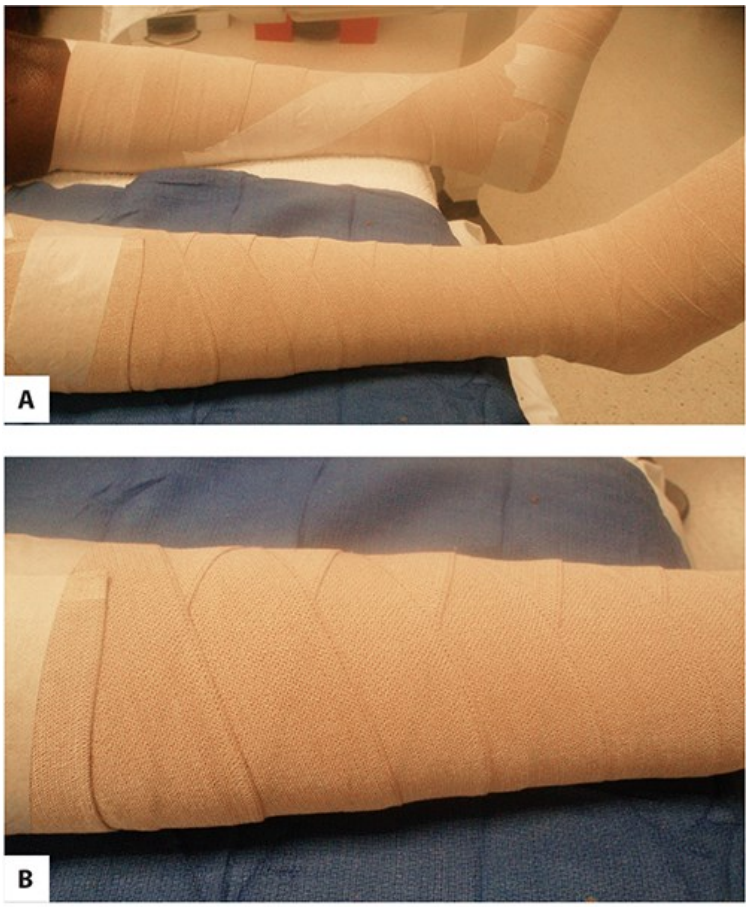

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FIGURE 4-69

Adding padding to a straight leg The ideal shape of the lower extremity to achieve the pressure differential for fluid return is a cone. **A.** If the leg is straight, or a pencil leg, padding around the calf helps to increase the calf circumference and thereby create an ankle/calf pressure gradient. **B.** The completed compression wrap illustrates the desired shape.

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FIGURE 4-70

Adding padding to a "champagne-bottle" ankle Similarly, if the ankle is atrophied and the calf enlarged, like the champagne bottle, padding around the ankle will decrease the pressure gradient so that it is closer to the ideal 40/18 mmHg. This can be accomplished by adding gauze padding, cast padding, or any soft conformable material around the ankle to create the ideal conical shape before applying the elastic layer.

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A number of compression garments or systems are available, including graded elastic compressive stockings, zinc oxide paste and gauze boots, multilayered compression systems, layered bandaging using either short-stretch or long-stretch bandages, and garments with adjustable elastic or non-elastic Velcro straps (**FIGURES 4-71** to **4-76**). Compression stockings are classified according to the amount of pressure provided at the ankle and are prescribed according to symptoms and history of ulceration (**TABLE 4-11**).

FIGURE 4-71

Graded compression garments A. Slippery toe sock. **B.** Graded compression garments for the lower extremities are available in knee high (primarily for CVI), thigh high, and panty style (both for lymphedema). There are also several types of donning aides, including the slippery toe sock seen in **A** and **B**, as well as a wire cage that is useful for patients who have difficulty reaching the foot.

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FIGURE 4-72

Zinc oxide paste and gauze boots For many years, the zinc oxide paste and gauze boots (commonly called the Unna boot) were the mainstay of compression for venous insufficiency. The gauze is impregnated with a zinc oxide paste that hardens after application, much like a cast, and forms a rigid compression bandage. **A.** The boot has been adapted in several ways, including using a hydrocolloid primary dressing over the wound and a self-adhering bandage on the outside (termed the *Duke boot*). **B.** The disadvantage of the zinc paste boots is the inability to contract as the extremity becomes smaller, thus providing less pressure and allowing the leg to telescope inside the bandage.

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FIGURE 4-73

Multilayer compression systems Multilayer compression systems include the following layers: (1) A soft cotton padding that is wrapped in a spiral. Additional layers can be placed over bony prominences (eg, the shin or ankles) to prevent pressure areas or around the calf or ankle to help shape the extremity. (2) A non-elastic layer that is wrapped in a spiral and provides the first layer of pressure. (3) A long-stretch layer that is wrapped in a figure-8 and provides the majority of the pressure. (4) A self-adhering bandage that anchors the system and prevents slippage. Layer 3 is omitted for modified compression if the ABI is between 0.6 and 0.8. The compression systems are changed every 3–7 days, depending on the amount of drainage from the wound and patient tolerance.

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FIGURE 4-74

Single-layer elastic bandages Single-layer elastic bandages are long-stretch and need to be used with caution. Application with consistent and even compression takes practice. Some brands have guides woven into the bandage to help the clinician monitor the amount of stretch, ergo tension, that is placed on the bandage. Studies have shown that single-layer bandages are not as effective in facilitating wound healing; they may, however, be helpful in reducing edema on extremities without wounds while compression garments are being obtained, provided the patient has adequate arterial flow.

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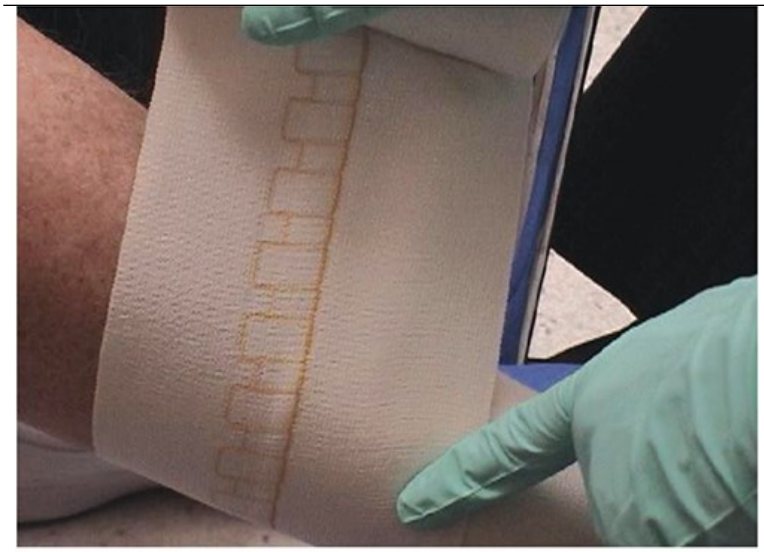

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FIGURE 4-75

Compression garments The FarrowWrap® (FarrowMed, Bryan, Texas) is a compression garment consisting of a foot piece and a series of Velcro bands that extend from the ankle to the upper calf, providing low resting and high working pressures like a short-stretch bandage system. The amount of tension can vary by the amount of stretch placed on each band. The system is beneficial for patients who have difficulty donning elastic garments. Used with permission from Rose Hamm.

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FIGURE 4-76

CircAid CircAid (CircAid® Medical Products, Whitsett, NC) consists of adjustable non-elastic Velcro bands that provide compression at recommended pressures for long-term edema management. These garments are custom fit to each patient and are available in styles for both wound healing and lymphedema.

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TABLE 4-11

Classification of Compression Stockings

Description	Amount of Pressure at the Ankle	Indications for Use
Support	15–20 mmHg	Early signs of CVI without ulceration, prophylaxis for high risk factors
Class I	20–30 mmHg	Signs of CVI without ulceration, post-sclerotherapy, prophylaxis for high risk factors, post-healing with inability to don/doff or tolerate higher compression, mild lymphedema
Class II	30–40 mmHg	Post-ulceration, pronounced varicose disease, moderate lymphedema, post-traumatic edema, burn scar management
Class III	40–50 mmHg	Severe lymphedema, severe CVI, with venous wounds and no arterial disease
Class IV	60+ mmHg	Severe lymphedema, elephantiasis, severe post-thrombotic disease

A review of the evidence and the above-mentioned guidelines provide direction on which compression is the most efficacious in promoting wound healing. For example, elastic compression is more effective than inelastic.[37] This may be explained by findings by Partsch, who measured sub-bandage pressure of zinc paste bandages immediately and 24 hours after application. They found that the pressure of zinc paste bandages *during dorsiflexion* dropped from 130–140 mmHg to 40–50 mmHg, suggesting that the effectiveness of a rigid system decreases because the leg reduces in size but the bandage does not.[53] Multilayer systems, on the other hand, "give" as the limb reduces in size and sub-bandage pressure remains high. Another difference is that inelastic bandages are more effective if the gastrocsoleus muscle is functioning—the muscle pushes against the rigid bandage with contraction and thereby produces pressure on the veins.[54] Elastic bandages provide compression both at rest and during ambulation, regardless of the muscle action, and are thus more effective for patients with a hypomobile or fused ankle or with paralysis. *Elastic bandages are contraindicated for patients with arterial insufficiency (and ABI 0.5 or less, or absolute ankle pressure less than 60 mmHg)*[55] *or post-bypass revascularization.*

An extensive Cochrane review concluded that wounds treated with compression heal faster than those treated without compression; multilayer systems facilitate faster healing than single-layer bandages; and multilayer systems with an elastic component were more effective than those without.[56]

Intermittent pneumatic compression pumps are useful in reducing edema at a first visit before applying a compression bandage, or for patients who

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do not tolerate other types of compression to use at home, especially after wound healing has occurred.[57] More discussion on pumps is included in Chapter 5.

The benefits of compression therapy have been well-defined, with significant improvement in pain, swelling, skin pigmentation, activity, and well-being as long as a high level of compliance with therapy is maintained. In fact, with a structured regimen of compression therapy 93% of patients with venous ulcers can achieve complete healing at a mean of 5.3 months.[58]

Treatment of Infection

Evaluating and treating a venous wound for infection or for periwound cellulitis is an integral part of caring for the patient and facilitating wound healing. Edema fluid neutralizes the fatty acids of sebum and inactivates the bactericidal properties of the skin; thus venous wounds are extremely susceptible to developing infection.[59]

The Wound Healing Society includes the following guidelines for infection control in the treatment of venous ulcers:

- Debridement of necrotic tissue on a venous wound reduces the bacteria and the risk of infection. Debridement is an integral part of wound bed preparation and facilitates the healing process.
- If the wound edges do not begin to epithelialize within 2 weeks of initiation of therapy, infection should be ruled out by either a tissue biopsy or quantitative swab culture for both aerobic and anaerobic bacteria.
- Infection is defined as $\geq 1 \times 10^6$ colony forming units (CFUs) per gram of tissue *or* any level of beta hemolytic streptococci and significantly impedes wound healing.
- Topical antimicrobial dressings are recommended for any wound with the above levels of bacteria; however, they should be discontinued when bacterial balance is achieved.
- Systemically administered antibiotics have not been shown to be effective in granulating venous wounds; however, periwound cellulitis should be treated with systemic gram-positive bactericidal antibiotics.[58]

Dressings

The amount of drainage and the presence of bacteria are the primary factors to consider when selecting a dressing. Initially, absorbent dressings such as alginates, hydrofibers, hydroactives, and foams are recommended to manage exudate and protect the periwound skin from maceration. As the wound granulates and drainage decreases, advanced dressings such as collagens may facilitate epithelial migration at the edges. Biological dressings or tissue and cellular therapies may facilitate closure once the wound bed is clean and granulated.[60] An extensive Cochrane review concluded that the dressing was not a factor in wound healing, and that indeed compression is the determinant intervention.[61]

Medications

Some oral agents (eg, diosmin and rutoside) in use in Europe have been reported to improve the feeling of heaviness, fatigue, and even edema of CVI, but these agents are not available in the United States. Pentoxifylline, an oral medication that improves the microcirculation and thereby improves healing rates, is supported by both guidelines and a Cochrane review.[62] Its benefit may be most noticeable in patients who have combined arterial and venous insufficiency. Additionally, if the wound is failing to show progress, a careful review of the patient's medications for drugs that may impede wound healing (eg, NSAIDs) is recommended.

Dysfunction in the calf and foot muscle pump plays a significant role in the pathophysiology of CVI, and graded exercise programs have been used in an effort to rehabilitate the muscle pump and improve symptoms.[34] Just as it is beneficial for prevention, exercise can improve dynamic muscle strength and calf muscle pump function in patients with all levels of CVI from varicose veins to venous ulceration (**TABLE 4-10**).

Adjunctive Therapy

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Venous wounds that decrease in size >40% in 4 weeks usually achieve full closure with standard care as described above. However, wounds that do not progress at this rate may benefit from adjunctive therapies, including electrical stimulation, non-contact low-frequency ultrasound, bilayered living cellular dressings (eg, Apligraf, Organogenesis, Inc., Canton, MA), ultraviolet C, and negative pressure wound therapy (for extensive wounds with some depth). Therapies that have not been shown to have statistically significant effects on venous wound healing include hyperbaric oxygen, laser, phototherapy, and whirlpool.[37,57]

Invasive Therapy

When CVI is refractory to medical and/or local treatment, invasive options are used to both reduce edema and facilitate wound healing. Removal of the saphenous vein with high ligation of the saphenofemoral junction has been the surgical standard for superficial saphenous insufficiency in more severe CEAP classes.[63] This procedure plus stripping of the great saphenous vein results in significant improvement in venous hemodynamics, provides symptomatic relief, and assists in ulcer healing.[64]

Advances in endovascular surgery have led to successful closure of the veins with incompetent valves using either radiofrequency or endovenous laser ablation. Both methods use a duplex-guided percutaneous access to the great or small saphenous vein. A tumescent anesthesia formula is administered along the course of the vein to be treated, which is then visualized with the duplex. Closure of the vein is accomplished with radiofrequency heat or laser.

These procedures are percutaneous, do not require general anesthesia, and are performed on an outpatient basis. Long-term follow-up studies have compared these procedures with conventional surgery and found equivalent efficacy and often reported improved quality of life.[65]

Surgical Interventions

The decision to recommend surgical intervention is based on the degree of disease according to CEAP classification and the success or failure of non-operative management. Listed below are the predominant surgical interventions and their indications.

Sclerotherapy

Sclerotherapy is a treatment for obliterating telangiectases, reticular veins, varicose veins, and saphenous segments with reflux. Rather than remove the diseased vessel surgically, sclerotherapy obliterates and seals the vessel in situ. Sclerosing, a procedure in which agents are injected into the venous segments by direct visualization or ultrasound guidance, can be used as primary treatment or in conjunction with surgical procedures for the correction of CVI.

Foam sclerotherapy is a relatively new procedure that combines traditional liquid sclerosing agents with gas to create a foam that has more surface area and expands to reach greater areas of the vessel. Emerging data suggest that it is safe and has equivalent healing and success rates with conventional surgery.[66] Recently, cyanoacrylate closure (CAC) has been used to treat saphenous vein reflux via a non-thermal, non-tumescent technique. Early results with CAC demonstrated similar closure rates and quality of life improvement as with radiofrequency ablation (RFA) over a 24-month study period.[67]

Endovenous Radiofrequency and Laser Ablation

Thermal energy in the form of radiofrequency or laser treatment is used to obliterate veins. This technique is used for saphenous vein reflux as an alternative to stripping and for its tributaries as an alternative to phlebectomy. These catheters generate heat, which causes thermal injury to the vein wall and thereby leads to thrombosis and eventually fibrosis. Several studies comparing endovenous ablation with conventional ligation and stripping found that the short-term efficacy and safety of ablation and surgery are comparable, although the surgical group had increased postoperative pain and bruising.[68]

Endovascular Therapy

Approximately 10–30% of patients with severe CVI have a significant abnormality in venous outflow involving iliac vein segments that contribute to the persistent symptoms.[62] Historically, iliac vein stenosis and obstruction causing CVI were treated with surgical procedures such as cross-femoral venous

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bypass or iliac vein reconstructions with prosthetic material. More recently, endovenous stenting has replaced the traditional bypass. In a large study of patients with CVI and evidence of outflow obstruction, iliac vein stenting resulted in clinical improvement with complete pain relief in 50% of the patients and complete resolution of edema in 33% of the patients.[49] The authors also noted that 55% of patients with venous ulcers achieved complete healing.

Open surgical procedures are reserved for patients who do not respond to less invasive treatment (usually with anatomic abnormalities) and form the basis for the endovascular therapies that are becoming widely used as a first-line intervention.

Ligation and Stripping and Venous Phlebectomy

Removal of the saphenous vein with high ligation of the saphenofemoral junction was one of the first treatments developed, going back to the mid-1800s or earlier.[69] This procedure has been the surgical standard for superficial saphenous insufficiency in CEAP clinical classes 2 to 6. Varicose clusters that communicate with the saphenous vein are often avulsed during the same procedure by phlebectomy.

In a study evaluating patients with venous ulcers and reflux of the superficial and deep venous systems, randomization to surgery (only on the superficial venous segments) plus compression therapy led to a reduction in ulcer recurrence at 12 months when compared with compression alone (12% versus 28%). This supported an additional benefit of correcting the incompetent superficial venous system for prevention of ulcer recurrence.[70]

Valve Reconstruction

Finally, venous valve reconstruction of the deep vein valves has been performed in selected patients with advanced CVI who have recurrent ulceration with severe and disabling symptoms. Venous valvuloplasty has been shown to provide 59% competency and 63% ulcer-free recurrence rates at 30 months.[62] A percutaneous valve has also been studied with moderate success, and the creation of a neovalve from the intima and media of the thickened venous wall to fashion a new monocuspid or bicuspid valve has also been undertaken with moderate success in select patients.[71]

CASE STUDY

CONCLUSION

Mrs. VB responded to standard care of compression, non-adherent dressings, and non-contact low-frequency ultrasound. The small wound on the left leg healed and the patient transitioned to a compression garment on that side. No vascular procedures were indicated due to her initial progress, including a decrease in pain levels to 5–6/10 with treatment.

Ankle exercises were included to increase range of motion and facilitate heel/toe gait sequence with good outcomes; the patient was able to ambulate without gait impairments; however, her work did require prolonged standing.

After 3 months of therapy, the wounds stalled (**FIGURE 4-77**). There were no signs of infection but because of the chronicity of the wounds, cultures were obtained and were negative. Negative pressure wound therapy was added to the standard care of selective debridement, absorbent dressings, and multilayer compression. The patient took a 3-week leave of absence from work with little improvement in both tissue quality and wound size. The patient had poor tolerance for negative pressure and requested that it be discontinued after a 3-week trial. The wounds did respond with increased granulation, flatter edges, and less epibole (**FIGURES 4-78** and **4-79**).

Collagen matrix dressings were added to the plan of care with slight increase in epithelial migration at the edges. At this point, the clinician reviewed again with the patient all of her recent laboratory values and medications. The patient admitted to taking 800 mg of Motrin a day, which she was advised to take for pain. After consulting with her physician, medications were adjusted to a different hypertensive medication given transdermally, and the patient discontinued Motrin with no need for alternative pain meds. Within 1 week new epidermis was visible at the edges, and the patient made significant and rapid improvement with full closure within 3 months.

FIGURE 4-77

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Right lateral lower extremity before use of negative pressure

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FIGURE 4-78

Right medial lower extremity after use of negative pressure

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FIGURE 4-79

Right lateral lower extremity after use of negative pressure

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In summary, current research suggests that endovenous therapy, RFA, and laser ablation are the gold standard. There is a select role for stripping and ligation but many of the historically used surgical techniques have been discontinued in favor of less invasive, less morbid endovascular techniques that offer equivalent benefit.

Post-Closure Care

Full re-epithelialization does not mean the wound is fully healed, only that it has entered the remodeling phase, and during this phase the skin is fragile and the wound is vulnerable to reoccur. Post-closure care to prevent recurrence of venous wounds includes consistent wearing of compression garments, continued exercise, and meticulous skin care. If a patient has a history of ulceration, at least Class II garments are recommended unless the patient is unable to don them or has confirmed PAD that has not been treated by a vascular specialist. There are several donning aids available for patients with other impairments (eg, arthritic hands or total hip replacement), and the patient needs to demonstrate competency in donning and doffing the garments before being discharged from therapy. For patients who require extensive compression therapy to achieve wound closure, transitioning to the garments may involve alternating use of the garments with weekly application of compression systems. This allows the skin to remodel and progress toward final healing, so there is less risk for abrasion or skin tearing with the use of garments, and it provides the clinician with the opportunity to monitor the patient compliance and response to the garments. Compression garments consisting of either elastic or non-elastic Velcro straps are also available for patients who have difficulty donning stockings. A recommended wearing schedule is to don the garments in the morning immediately upon awakening and before the limb swells, and to remove them at night before showering and providing skin care. The importance of adherence to post-healing protocols cannot be overemphasized to patients who have healed venous wounds.

SUMMARY

Venous wounds result from chronic venous insufficiency and are a common and difficult condition to manage. Venous insufficiency can be primary, secondary, or congenital. Mechanisms for the failure of the venous system are valve incompetence, obstruction or post-thrombotic syndrome, inherent vessel wall factors, and impaired calf-pump mechanism. Venous disease causes integumentary changes visible on physical examination; it is usually diagnosed with non-invasive means and classified according to universal consensus guidelines. Compression therapy is the main component of treatment, with topical/local wound care, exercise, and adjunctive therapies used to facilitate healing. Endovascular and/or surgical treatment may be required in the most severe cases.

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